

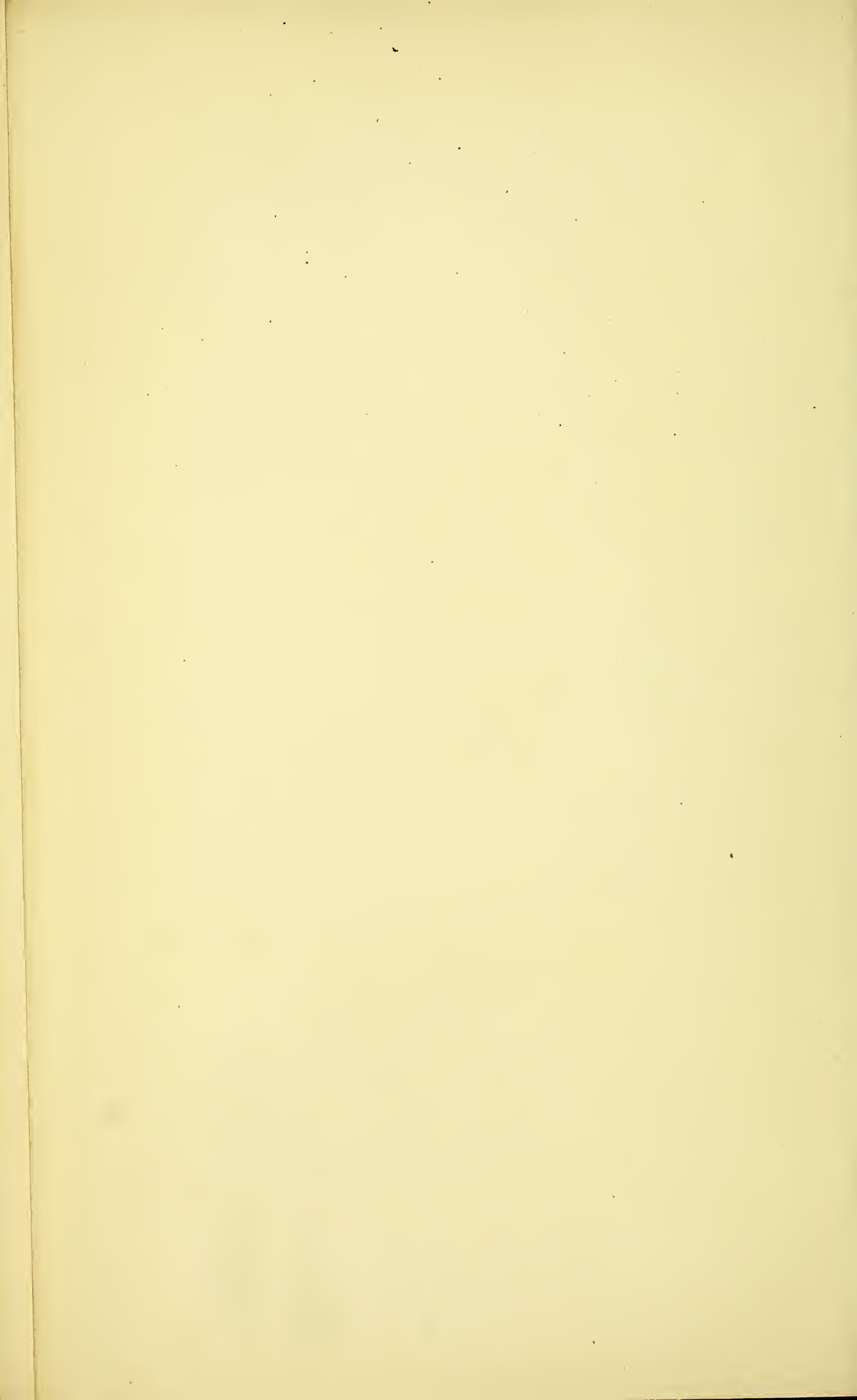




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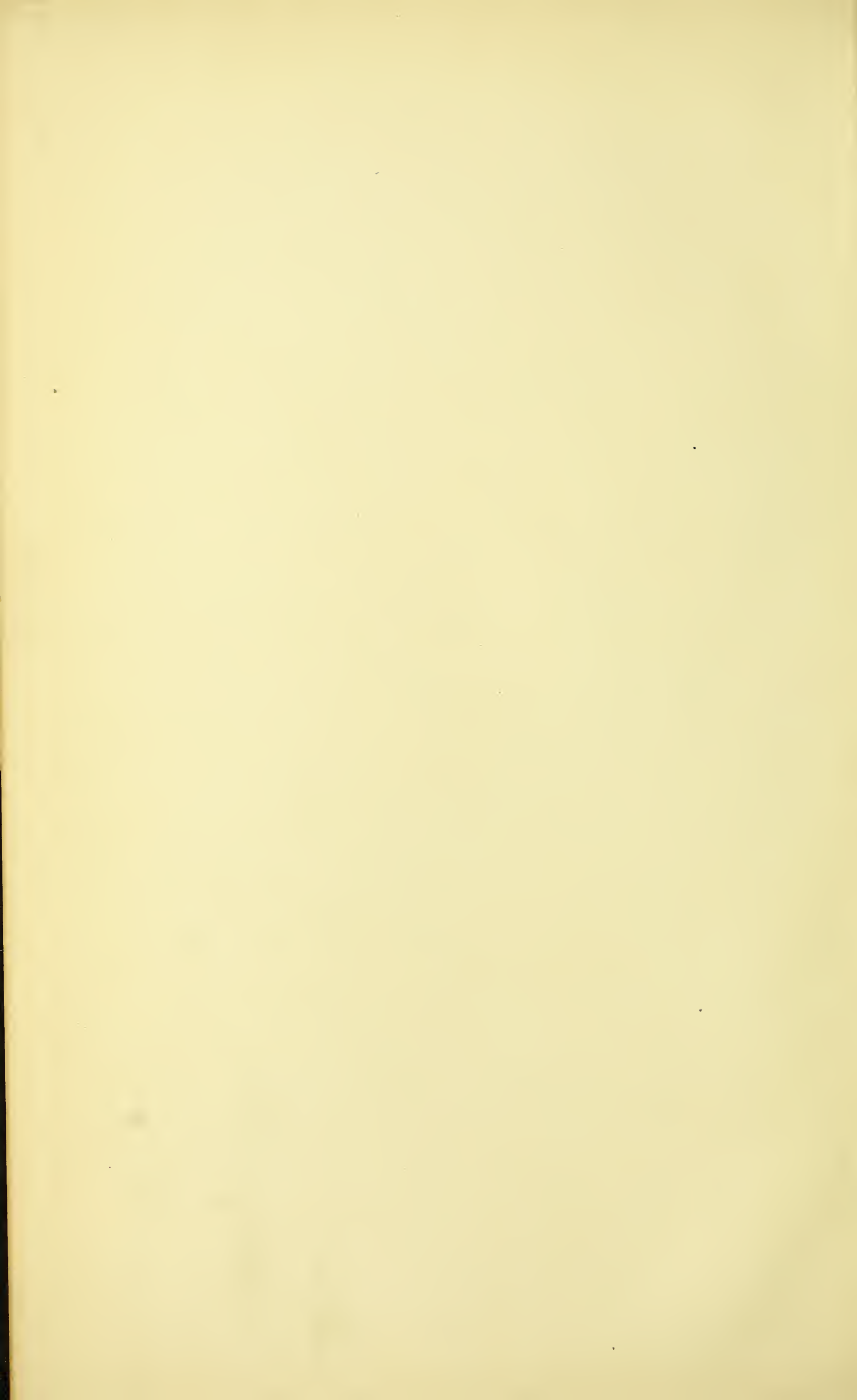




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# ENCYCLOPÆDIA MEDICA

UNDER THE GENERAL EDITORSHIP OF  
CHALMERS WATSON, M.B., M.R.C.P.E.

VOLUME IX

*OSTEO-ARTHROPATHIES to PREGNANCY*  
*(PHYSIOLOGY)*

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**Orchitis.** *See* TESTICLE.

**Osteo-Arthritis.** *See* RHEUMATOID ARTHRITIS.

**Osteo-Arthropathies.**

OSTEOPATHIES . . . . .	1		PULMONARY OSTEO-ARTHROPATHY	4
ARTHROPATHIES . . . . .	2			

THE relationship between disorders of bones and affections of the nervous system was noted by Mitchell in 1831, but the condition was little studied until Charcot drew attention to it some years later.

Trophic disorders, both of bones and joints, occur in varying degrees in several diseases of the nervous system, but notably in the course of locomotor ataxy, and the lesions occurring in this affection may be conveniently described as a general type of the disorder.

The bones and joints may suffer separately or together, and accordingly are termed osteopathies, arthropathies, and osteo-arthropathies.

OSTEOPATHIES.—In locomotor ataxy changes frequently take place in the nutrition of the bones which cause them to fracture with the very slightest mechanical violence or even spontaneously.

The fracture is usually quite painless, but is often accompanied by considerable œdema of the neighbouring tissues; occasionally lightning pains appear to be specially prevalent in the region before the bone fractures, but otherwise there are no premonitory symptoms. Union, which generally takes place with the usual rapidity of other fractures, is often accompanied by an excessive amount of callus—a fact which has been explained by the mechanical friction to which the painless ends of the fracture are apt to be subjected; marked deformity of the limb, due to displacement of the bones, is also another common sequel. The lower limbs are more frequently fractured than the upper, and women appear to suffer more often than men (Marie).

*Morbid Anatomy.*—The bones are lighter than normal; to the naked eye they often have a worm-eaten appearance, and on section the medullary canal is found to be increased in size at the expense of the bone around it. There may be some irregular deposits of new bone taking place at the same time.

Microscopically the Haversian canals are dilated, and absorption of bone is proceeding in their vicinity. Regnard has shown by chemical analysis that there is a decrease in the inorganic constituents of the bone, especially of the lime salts, and an increase in the organic constituents, especially fat.



ARTHROPATHIES.—Trophic lesions of the joints as they occur in locomotor ataxy were first described by Charcot, and hence they are frequently spoken of as “Charcot’s joints.”

The onset of tabetic arthropathies is usually acute; the joint becomes distended with fluid in a few hours, and generally, though by no means constantly, there is no pain and no fever. The distension of the joint is often accompanied by swelling of the parts around, and movements of the limb are much impaired. In other instances the onset is more gradual, and extensive disorganisation of the joint may take place without the occurrence of any acute symptoms.

The fluid tends to subside in a few days or many weeks, and, in disappearing, it leaves very various conditions behind it; in the more favourable cases the joint appears to have suffered but little, but in others the articular surfaces are partially or wholly destroyed.

The liability of the different joints to be attacked varies considerably; Charcot placed their frequency in the order of knee, shoulder, elbow, hip, and wrist, but subsequent statistics show that the hip-joint must be given a considerably higher place in the list. The small joints of the hand are but seldom affected.

Clinically an atrophic and a hypertrophic form can be recognised. In the atrophic form atrophy of the bones is the predominating feature, while in the hypertrophic variety new deposits of bone take place round the joint as in rheumatoid arthritis. As was shown by Kredel, the atrophic form is more commonly found in the ball and socket joints (hip and shoulder), and the hypertrophic in the hinge joints (knee and elbow).

*Morbid Anatomy.*—The fluid which distends the joint is usually clear; occasionally it may contain blood, especially if the arthropathy is complicated by an intracapsular fracture, which is not a very uncommon occurrence; suppuration never occurs unless, of course, the joint is in some way accidentally infected from without. In cases of any standing the articular surfaces are greatly modified. In the atrophic form the head of the bone often almost or entirely disappears, and, the articular cavity undergoing similar changes, all formation of the joint becomes lost; in the hypertrophic form the articular cartilage disappears, leaving underneath porous cancellous bone (only rarely is it eburnated, as in rheumatoid arthritis), while at the margins of the joint there are prominent irregular bony outgrowths.

The ligaments and capsule of the joints are lax, so that an abnormally free movement of the joint can be obtained, and the internal ligaments frequently share in the destructive process and disappear. In some cases the irregular ossification round the joints extends to the capsule and external ligaments. The synovial membrane is thickened and adherent to parts around; it may be studded with villous outgrowths, and in many cases it is partially destroyed. If an intra-articular fracture exists, as already mentioned may be the case, the condition is further complicated by the presence of loose or partially detached fragments of bone within the joint.

OSTEO-ARTHROPATHIES.—The coexistence of bone and joint lesions is well exemplified by the lesions of the vertebral column as they occur in locomotor ataxy, and also better still by the “tabetic foot.”

The vertebral lesions are rare; they are characterised by spontaneous fracture of the bodies of one or more vertebræ associated at the same time with changes in the intervertebral articulations. The lumbar region of the spine is the part most often affected, and a painless angular curvature develops, which, however, but seldom damages the spinal cord.

The tabetic foot varies in its shape according to the different displacements



of the tarsal bones. One of the commonest forms is the "flat foot," with thickening on the dorsum or instep, as described by Charcot; in other cases the long axis of the foot deviates outwards or inwards; sometimes the arch of the foot is raised. In a case recorded by Lunn there was great deformity of the plantar arch, the tarsal bones in front of the astragalus standing out very prominently on the dorsum of each foot. Sometimes the deformity is further complicated by an arthropathy of the ankle-joint, and as Targett has well shown, no form of dislocation can be considered peculiar to the tabetic foot.

The changes in the bones of the tabetic foot belong to the atrophic type, the bones are light and porous, with little tendency to fresh deposits, and their articular cartilages gradually disappear as in the larger joints. The presence of a perforating ulcer may greatly modify the appearances of the bones by the secondary infection which it is apt to set up.

*Arthropathies in Syringomyelia.*—In syringomyelia changes occur in the joints which are almost identical with those already described in tabes, but they generally run a more chronic course, and the upper limbs are more liable to be affected than the lower.

*Mode of Production of Osteo-Arthropathies.*—It is now generally conceded that osteo-arthropathies occurring in locomotor ataxy and syringomyelia are directly dependent upon the lesions of the nervous system.

Volkman's idea that the joint affections in tabes were due to traumatism, owing to the irregular movements of the limbs, became untenable when it was known that the joints were frequently affected in the pre-ataxic stage of the disease; likewise Strümpell's theory that the lesions were syphilitic fell to the ground when their prevalence in syringomyelia, which has no special syphilitic antecedents, became an acknowledged fact. The establishment of "Charcot's joints" as a distinct disease apart from rheumatoid arthritis caused some hesitation, but a careful comparison soon made it clear that Charcot's claim for this separation was justified. The main point in which the two diseases differ are in distribution, mode of onset, presence of pain, and in the rapidity with which a joint may be destroyed. Tabetic arthropathies tend to affect single joints, while rheumatoid arthritis tends to affect several small joints. A Committee of the Clinical Society of London, appointed to examine this question, found that in sixty-six cases of tabetic arthropathy a single joint only was affected in forty-one; in nineteen cases two joints were affected, and in only six were more than two diseased.

The mode of onset of Charcot's joints is acute, painless, and rapid, often leading to advanced disorganisation of the joint within a few weeks; in rheumatoid arthritis the onset and progress are nearly always both gradual and accompanied by pain. The morbid anatomy of the two affections is in some cases closely alike in its appearances, but the eburnation of bone beneath the corroded articular cartilages, which is generally so prominent a feature in rheumatoid arthritis, is nearly always absent in tabetic arthropathy, the bone in the latter being porous and devoid of any sclerosis beneath its cartilages.

As to the exact way in which diseases of the nervous system produce trophic changes in bones and joints there is but little known.

The fact, however, that locomotor ataxy is the condition with which the lesions in question are most commonly associated, suggests at once that it is the sensory fibres which are primarily at fault, and this is borne out by the morbid anatomy of syringomyelia, and further narrowed by the facts that in the latter case the sensations for pain and temperature are frequently the



only ones affected, and also that the nerve roots and peripheral fibres are not involved as they are in tabes.

As to how lesions of the sensory fibres modify the nutrition of the bones and joints nothing definite is known; it may be that there are special trophic nerves associated with them, or the changes may possibly be produced by the vasomotor fibres; the motor cells of the anterior cornua probably take no important part in the matter, since no lesions comparable to those with which we are now concerned occur when these cells are definitely diseased as, for instance, in infantile paralysis and progressive muscular atrophy. Pitres and Vaillard found changes in the nutritive nerve of the bone, but it seems fairly obvious that lesions of peripheral nerves cannot be considered in any sense a constant cause, since in a disease so common as peripheral neuritis lesions of joints comparable in any way to those of locomotor ataxy or syringomyelia are exceedingly rare.

Trophic lesions of bones and joints, as already stated, occur to some extent in other diseases than those already described.

In leprosy osteo-arthropathies have been described by Heiberg which have many of the characteristics of the osteo-arthropathies of tabes, viz. swelling and laxness of the joint capsules with wearing away and atrophy of the ends of the bones, and the same observer has also discovered a condition which closely resembles the "tabetic foot."

In general paralysis of the insane the bones become brittle and are easily broken, but it is very doubtful whether the changes can be put down to any direct trophic influence; true arthropathies, when present, are probably of tabetic origin, since the relationship between tabes and general paralysis is such a close one.

In infantile paralysis the chief characteristic is arrested growth of bones.

In peripheral neuritis chronic changes may take place in and around the joints, which lead to thickening and occasionally to ankylosis, but—with the possible exception of those occurring in leprosy which may be of peripheral origin—they are not in any degree comparable to the lesions of tabes and syringomyelia.

Arthropathies have occasionally been described as a result of hemiplegia, and they may also sometimes be found as a complication of Pott's disease and injuries of the spine.

**PULMONARY OSTEO-ARTHROPATHY.**—Changes in the bones and joints are frequently found in cases of pulmonary tuberculosis and other chronic chest affections, such as empyema, bronchiectasis, and abscess of the lung. Marie first drew attention to these conditions in the course of his writings on acromegaly. In a typical case the fingers are greatly enlarged, especially at the last phalanges which are very much swollen, the heads of the metacarpal bones are slightly enlarged, and also the wrists. The feet may be enlarged in a similar manner.

The significance of these changes is at present doubtful. Godlee considers that in many instances there is no enlargement of the bones at all, basing his conclusions upon Röntgen ray photographs and also on the fact that patients sometimes rapidly recover when their chest complaint is cured. In other cases erosion of articular cartilages and subperiosteal thickening of the bones have been found, but here Thorburn has shown that such changes are probably often due to local tuberculosis.

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## **Osteomalacia** (MOLLITIES OSSIUM).

See PREGNANCY, PATHOLOGY OF; also LABOUR, CONTRACTED PELVIS.

THOUGH known from early times, osteomalacia was distinguished from rickets only in the year 1772; when it was recognised that while the one disease prevents the deposition of mineral matter in the bones during their development, the other softens them by the removal, first, of inorganic, and, later, of organic matter during adult life. Osteomalacia is typically seen in certain districts, in the half-starved woman with a large family, who works in the fields exposed to wet and cold, suckling one child and heavy with another.

The first symptom, as a rule, is pain in the sacrum, spine, and pelvic bones, which later involves the legs and sometimes the arms. The pain is continuous, but is increased by pressure and by movement. Difficulty in walking is next observed, and owing to inability to flex the thigh on the abdomen, the patient adopts a waddling gait. Increased knee-jerk, ankle clonus, muscular tremors, inability to stand with the eyes shut, and various other nervous symptoms, are early features of the disease. Muscular weakness is of general occurrence. As the disease advances certain bones become softened owing to the removal of their inorganic portions, and bending occurs which is determined by purely mechanical principles.

The pelvis first shows deformity. The weight of the body, acting through the sacrum, and the resistance of the ground, transmitted through the acetabula, push in the pelvis, behind and laterally, and so produce the “beaked,” “rostrate,” or “triradiate” pelvis, which is figured and described, together with its effects upon labour, in all obstetric works (see “Contracted Pelvis”). The spine may become twisted in various ways, so greatly reducing the height of the patient. Softening of the ribs causes deformity of the chest, as the weight of the arms pushes its sides together, throwing forward the sternum. The angles of the scapulæ may become incurved; and, lastly, the long bones may be affected, and are occasionally fractured and bent. The other systems are as a rule unaffected. Recovery may occur spontaneously even after many years, or the condition may progress until the patient becomes bed-ridden and dies of concurrent disease.

The nervous system is found post-mortem to be free from gross lesions. Histopathological examination has, however, revealed organic changes in the cord which appear to begin in the cells of the anterior cornua. These are not inflammatory in character, and clinical evidence shows that they are capable of repair. There is early congestion of the periosteum with dilatation and congestion of the medullary vessels. The medullary canals are enlarged as are the cancellous spaces. The inorganic matter is removed from the areas surrounding the Haversian canals, and absorption of the organic portion of the bone may follow; osteoclasts are seen in the absorbed areas, as in the physiological process of bone removal. Repair may be observed going on side by side with absorption.

Whilst the proportion of inorganic to organic matter in normal bone is about 2 to 1, this is reversed in osteomalacic bone till the proportion is as 1 to 2. The weakened muscles show, post-mortem, fatty degeneration,



multiplication of nuclei, and other changes similar to those observed in progressive muscular atrophy. During the active phases of the disease phosphates abound in the urine; but apart from this it cannot be said that examination of the urine or of the blood has had results which are consistent or valuable.

Osteomalacia is much commoner in women than in men, though numerous cases of its occurrence in male subjects are recorded. The disease most frequently occurs during the period of sexual activity, though in several cases it has appeared after the menopause, and it is also said to have been observed in childhood. Pregnancy and lactation, especially if frequent, favour the advance of the disease and cause exacerbations. About 74 per cent of the recorded cases have occurred in connection with pregnancy, the remainder being divided between nulliparous women and men. The disease is so rare in most parts of the world that it is considered as endemic in portions of Italy and Switzerland, in Bavaria, Wurtemberg, Baden, and Alsace, where it is frequently observed.

So many theories are still in vogue as to the cause of osteomalacia, that it is clear that we have no real knowledge of the matter. The view which explains the greatest number of the features of the disease is that it is a primary affection of the nerve cells of the cord. The nervous symptoms have been regarded as due to pressure consequent on deformity, but in reality they appear before there is any change in the bones. Charcot's disease forms a suggestive parallel. Nervous diseases are markedly hereditary, which accounts for the distribution of osteomalacia better than suppositions as to food and water. Indeed starvation, exposure, frequent pregnancy, and prolonged lactation are of so universal distribution that it is impossible to regard them as causes of a disease so restricted to certain localities. As secondary causes, given a primary and inherited nervous lesion, they may well act in making manifest a latent weakness.

The early diagnosis of osteomalacia is not possible. Most cases are considered either as rheumatism or as spinal disease until deformity appears. The so-called *osteomalacia carcinomatosa* is the only condition with which advanced cases may be confused, and this is usually distinguished by other evidences of malignant disease, primary or secondary. Assuming that the primary lesion of osteomalacia is a spinal one, an early provisional diagnosis of spinal disease would of course be correct, and would be made more definite on the appearance of deformity. The prognosis is now very much better than was the case a few years ago. Hygienic surroundings, cod-liver oil, phosphorus, bone-marrow, and salt baths have all given excellent results. The idea that removal of the ovaries cures the disease has arisen partly as a corollary to the theory that reflex irritation from the reproductive organs is its cause. The numerous recoveries which have occurred after Porro's operation (done to secure delivery at term in cases of advanced deformity) have suggested the adoption of double oophorectomy (sometimes accompanied by removal of the uterus) for the cure of the disease in non-pregnant women.

As, however, numerous women (and men) have recovered under good surroundings and internal medication, these should always be patiently tried, and should be continued even after operation, should this be thought necessary; for many cases of relapse after oophorectomy have ultimately recovered under medical treatment. For the course which should be followed when a sufferer from osteomalacia is pregnant, the reader is referred to the discussion of the subject under the heading "Contracted Pelvis."



**Osteo-Myelitis.** See BONES, DISEASES OF (vol. i.).

**Osteotomy.** See DEFORMITIES.

**Otorrhœa** is a symptom of disease in the outer or middle ear and its annexa. It is most commonly dependent on an acute or chronic inflammation in the tympanum—conditions described in an earlier part of this work (vol. iii.). The present article deals with the symptoms *per se*, and the manner in which it has to be studied and treated.

Under normal conditions the bacteria present in the outer and middle ear exist as saprophytes and have no pathogenic significance, but it is otherwise in diseased states of the ear and associated mucous membranes of the Eustachian tube and naso-pharynx, in which we have circumstances favourable to their further development and activity. The diseases of the outer ear attended by otorrhœa can be shortly dismissed. It may be dependent on the presence of a foreign body in the meatus; in investigating this point, care must be exercised to determine the presence of any small foreign body far in the meatus next the membrane at its anterior part. Or it may be due to an inflammatory condition of the cutaneous lining membrane, such as is present in cases of eczema or parasitic otitis externa (otomycosis). In yet other instances it may be traumatic in origin. But it matters not which of these conditions has been the starting-point of the disorder; the important point to bear in mind is that in every case we are dealing with a local bacterial infection. Bearing this in mind, the treatment is simple and calls for little comment. The removal of any foreign body must be effected; the meatus should be syringed with a non-irritating antiseptic solution, with a frequency proportionate to the severity of the case, and thereafter the meatus should be carefully dried with a pledget of antiseptic cotton wool. This is best done by the use of a wooden match, the end of which is encircled by the wool and inserted carefully into the ear. Dryness is inimical to bacterial growth. In bad cases it may be desirable to counter-irritate the diseased surface by silver nitrate  $\frac{10 \text{ to } 30}{\text{grs. to } \frac{1}{3}}$  or other stimulant, with the object of inducing a more favourable local reaction. No further local measures are called for.

The cases dependent on chronic disease in the middle ear are more serious, and correspondingly more difficult to treat. The clinical history of these patients is usually perfectly clear, and leaves no room for difficulty in the diagnosis; and while in the majority the course of the otorrhœa is a simple one, and the treatment equally simple and satisfactory, in other cases the reverse obtains, and the treatment is very unsatisfactory. It is the latter group that is specially interesting. What is the explanation of those cases of chronic suppuration which fail to respond to the ordinary treatment, comprised in thorough cleanliness, asepsis, and it may be a little judicious stimulation? Is there any special bacteriology of these cases? Is there any particular microbe or combination of microbes which finds a particularly suitable nidus in the middle ear or in the pus itself of these cases? These are questions that can hardly be answered; but they should be kept in mind and, when opportunity affords, investigated. We must, of course, bear in mind that in cases of suppuration, mainly in the attic (*v.* vol. iii.), the difficulties of suitable drainage are considerable, and this mechanical difficulty may in itself be the sole cause of the long continuance of the discharge. But this in no way accounts for all cases. A case that was under the writer's observation for over three



years illustrates this point well. When first seen, the child, æt. 21 months, suffered from an otorrhœa of some months' standing. The persistent use of boric lotion thrice daily, carried out carefully by the mother, was not followed by any marked improvement. The continuance of the discharge led to an operation on the mastoid, at which the diseased parts were freely exposed and scraped, and subsequently treated in the hospital. The conditions persisted, and later the naso-pharynx was scraped, as it was thought possible that a slight cushion of adenoid tissue might be aggravating the middle ear disease. No improvement resulted; the free discharge persisted. This case was again operated on, more than a year later, by another surgeon, and still the patient's state was apparently uninfluenced. A third operation was attended by a like result. The surprising thing about the case was the relatively slight influence which this long-continued free suppuration seemed to exert on the patient. The child grew and appeared to thrive in a really remarkable degree, and when  $3\frac{1}{2}$  years old met with a very serious burning accident, involving the skin of nearly one-third of the body. Curious to relate, this very serious accident seemed to affect this child even less than it would have done a robust child. This fact was so striking that one could not but wonder whether the previous suppuration had exerted anything of the nature of an immunising effect on the tissues of the patient. When the patient again came under the writer's observation six months later, a bacteriological examination of the pus was made, with the following result. The pus showed a few round cocci which stained by Gram's method, but the most striking micro-organism was a short, straight, *actively* motile bacillus, which stained homogeneously, formed no spores, and was decolorised by Gram's method. These were present in great abundance. On an agar plate they formed greyish-white colonies of moderate size, somewhat flat in character, with a sharp clear margin and dark granular centre. The bacillus rendered bouillon diffusely cloudy; it formed gas in a glucose agar medium and liquefied gelatine by the second day. This last point served to differentiate it from the bacillus coli, which it otherwise closely resembled in many respects. The blood serum of this patient did not agglutinate or interfere with the motility of this bacillus after twenty-four hours in a dilution of 1 to 30. The last-mentioned point seems, at first sight, to suggest that this bacillus bore no causal relationship, but the data are insufficient to dogmatise on this point, as it is doubtful if the serum necessarily acquires agglutinating properties in cases of a purely local suppuration. It may have been that this bacillus was an accidental contamination. It should perhaps be stated that this microbe was apparently not one of the special micro-organisms referred to in the section on the ear (vol. iii.). These points are mentioned with the object of emphasizing the importance of a bacteriological examination in all cases of an intractable nature. A more extended knowledge of the bacteriology of these cases would help much to elucidate their etiology.

With regard to treatment of these cases a few words will suffice. The line of treatment is essentially the same as already indicated for the external ear discharge, only more frequent syringing and even greater care in drying the accessible parts of the ear are called for. A few cases call for operative interference. These have been discussed in vol. iii. But there is one line of treatment which is valuable, and merits probably greater attention than it has yet received. We refer here to an adequate and yet not excessive stimulation of the tympanic mucous membrane by the local application of stimulating lotions, *e.g.* 10 to 15



drops of a solution of equal parts of glycerine and rectified spirits. Aniline oil in rectified spirits has also been used with marked success in some cases, but the glycerine and spirits is probably, on the whole, more advisable, as toxic effects have been known to follow the administration of aniline oil. These remedies doubtless exert their influence through the vascular reaction induced by them and the resulting greater power of resistance associated with a more vitally active mucous membrane. It must not be forgotten that treatment directed to the Eustachian tube, more especially the use of the catheter, is very helpful in some of these cases.

### Ovaries, Diseases of the.

THE anatomy of the ovaries is described under "Generation, Female Organs of," and a special article is devoted to the "Broad Ligament, Diseases of." *Cystic Tumours of the Broad Ligament* being closely allied to ovarian cysts will be included in the succeeding paragraphs.

Physiology, p. 9.

Removal of ovaries for cure of cancer of breast, p. 10.

Malformations, p. 11.

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Hernia and allied affections, p. 12.

Hyperæmia, p. 13.

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Inflammation—Tubo-ovarian cysts, p. 13.

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Tumours of ovary.—The Pedicle—Capsules—Complications associated with tumour—Symptoms—Diagnosis—Treatment—Ovariectomy—Complications following ovariectomy, p. 15.

Tumours of ovarian ligament, p. 32.

Tumours of broad ligament, including primary dermoid, p. 33.

Primary ovarian pregnancy, p. 36.

PHYSIOLOGY OF THE OVARY.—It has long been known that the ovaries produce ova, and are closely associated with the general sexual characteristics of the female subject. It now appears more than probable that these characteristics are influenced not directly by ovulation, but rather by some product or secretion elaborated by the ovary.

An infant bears thousands of ova in the ovaries at birth, and the majority undergo premature involution before puberty. Then begins the process of ovulation and menstruation, and the capability of impregnation and gestation follow. The precise relation of these processes to each other has never been strictly determined, and demands more lengthy discussion than is at our disposal. An ovum after impregnation can develop a human being with tissues in their right places; it is just possible, then, that the phenomenon of ovarian dermoid means that an ovum not impregnated can under some unknown influence develop the tissues of the human body, but cannot, so to speak, arrange them. The evolution of the ovum and the involution of the ovisac into a corpus luteum are well known. These active changes slowly disappear during the menopause.

Absence or extreme arrest in development of the ovaries involves sterility, with distinct but not very definite modifications in general sexual characteristics. Destructive disease of the ovaries, such as the inflammation and interstitial hæmorrhages which occur during certain exanthemata, have been known to produce the same effects. The results of alleged complete removal of the ovaries by operation show less definite results. As a rule,



sterility and all the symptoms of the climacteric follow, with speedy or gradual disappearance of the sexual appetite. But in other cases sterility is the sole consequence, the period persisting, the sexual appetite is often increased, or even develops when the patient was previously frigid. Uterine fibroids, especially when limited to the upper part of the uterus, often decrease in size after removal of the ovaries, but sometimes remain quite unaffected and grow larger. Clinical records of removal of the ovaries must, however, be accepted with great reserve, particularly in relation to physiology. For it is not always certain that all ovarian tissue is removed at the operation. This tissue often runs along the ovarian ligament close up to the uterus, so that some is left behind when the tube is amputated, unless the pedicle be cut dangerously short—a surgical error which the operator instinctively avoids.

There can be no doubt that the ovary eliminates some product which has a general effect on the economy. "Ovarian oöphotherapy" has, however, as yet given very uncertain results. Dalché, whose reports seem the most trustworthy, gives cachets consisting of a grain and a half of fresh ovarian tissue from the sheep. One or two are given daily at first; three are, as a rule, quite enough later on. His results are not very definite; thus he cured a chlorotic young work-girl of anæmia, but amenorrhœa, which had not existed before, set in with the treatment. The phosphates in the urine were greatly increased; this is interesting, as removal of the ovaries arrests mollities ostium and checks the elimination of phosphates. In Fosbery's case climacteric flushing was successfully treated by 5-grain palatinoids of ovarian gland given three times daily; Dalché claims similar experiences. Martin (*loc. cit.*) and Kippenberg report that fourteen out of thirty cases of double ovariectomy were "benefited" by this treatment, but they remain sceptical, and admit that the extract often causes dyspepsia, and that the "cure" may be by suggestion.<sup>1</sup> Saalfeld has made use of ovarian juice for skin diseases during the menopause, and also for prurigo at the menstrual period with success.

REMOVAL OF OVARIES FOR THE CURE OF CANCER OF THE BREAST was first practised by Beatson of Glasgow in 1895, and he made public his results before the Edinburgh Medico-Chirurgical Society in May 1896. He believed that some pathological condition of the ovaries was the exciting cause of cancer of the breast. There is a close relation, he noted, between the ovaries and the secretion of milk; epithelial proliferation in a healthy breast becomes a factor in milk production, whilst in an unhealthy breast it becomes a factor in developing cancer. Removal of the ovaries appeared, he maintained, to have an inhibitory influence on malignant proliferation of epithelial cells. Since 1896 Watson Cheyne, Herman, and particularly Stanley Boyd, have tried this practice and published their results. Beatson, with whom Herman agrees, advocates the administration of thyroid extract after oöphorectomy as a powerful lymphatic stimulant. Stanley Boyd has observed the effects of the extract in several cases where oöphorectomy was also practised, and in others where the ovaries were not removed, and came to the conclusion that its action on the disease is very doubtful. In Page and Bishop's case of recurrent cancer of the breast the extract was given for a year without effect; then the patient disappeared, but continued taking the drug. When she was again seen six months later the disease had disappeared. But Boyd questions whether the result was due to the

<sup>1</sup> There is no evidence that any of the fourteen would have suffered from the loss of both ovaries. Ferré and Bastian find that a glycerine and water extract acts physiologically on a male more powerfully than on a woman, producing orgasm followed by paralytic symptoms.



thyroid extract, and quotes Gould's case of spontaneous disappearance of advanced mammary cancer without any kind of treatment. From 3 to 20 grains of the thyroid extract were given daily in the above cases. Oöphorectomy has certainly proved of temporary benefit in many cases of recurrent or inoperable cancer. In the summer of 1900 Stanley Boyd gave details of thirty-eight cases, in seventeen of which considerable improvement had resulted. He found that the operation was practically useless after the menopause, and admits that a great deal more experience will be necessary to determine its mode of action and range of benefit.

ACCESSORY OVARIES.—The term "accessory ovary" is usually applied to a small lobe of ovarian tissue attached to the normal ovary by a pedicle. In an example under the author's own observation there was on the opposite side a fibro-myoma of the ovarian ligament half an inch in diameter. De Sinéty once detected a supernumerary ovary which bore on its surface ciliated, not columnar epithelium, and contained cysts lined with the same kind of cell and bearing ova. About 4 per cent of post-mortem subjects show these insignificant "accessory ovaries" (Beigel and von Winckel). Portions of adrenal tissue occasionally found in the broad ligament must not be placed under the present heading, which also must not include instances when an ovary is completely divided into two parts by a groove. A few examples of an absolutely distinct third ovary have been reported by writers of repute. In von Winckel's, the best known, the third ovary is represented as springing from the front of the uterus immediately internal to the right round ligament. Schultz-Schultzenstein, in figuring an intraligamentary myo-sarcoma from a post-mortem subject where both normal ovaries existed, represents the tumour springing from a pedicle anterior to the Fallopian tube—in the same position, in fact, as in von Winckel's case; but his belief that it was a sarcoma of a third ovary is based on uncertain grounds. Pedunculated solid tumours of the broad ligament quite independent of the ovary and uterus are not very rare. In Winkler's case two left ovaries of normal size existed; in Keppler's two such ovaries were provided with a Fallopian tube apiece. One or two other more doubtful examples of the anomaly are recorded. When menstruation continues after alleged removal of both ovaries we must not be too ready to assume that an accessory ovary has been left behind. In such a theory there are many sources of fallacy.

ABSENCE OF OVARY.—This condition is either teratological, or simply means that the organ has become adherent to some other structure, and has separated from its normal connections, so that it exists but away from its normal place and attachments. Assumptions about the ovary in any case of malformation of the genitals are unsatisfactory. Both ovaries may be present when the uterus is represented by a mere cord (Cruise). Teratological absence of the ovaries has been found in every degree. Kossmann and others have proved on clinical evidence that total absence not only of the ovaries, but also of the uterus, is not incompatible with survival to adult life. But the malformation is often associated with more or less mental disease, with frigidity, or in some cases, it would appear, with nymphomania. Indeed, it seems as though it were sometimes the cause of the psychical derangement. In a case of a female lunatic, who died aged thirty-eight, and whose insanity began at puberty, the author found a uterus foetalis with atresia of the os externum, whilst the ovaries had atrophied or rather never developed. Painful coitus and other distressing symptoms are observed in cases of absence of the ovaries. *Absence of one ovary* is a teratological condition rarely though occasionally co-existent with perfect



development of the uterus and opposite ovary. Scanzoni found it associated in two cases with arrested development of the corresponding mammary gland. *Rudimentary ovaries* come under the present category, but are more frequent than the above malformations. They are often associated with similar grave psychical complications. Not rarely they give rise to severe pain, for which reason they have been successfully removed.

*Absence of the ovary from its normal side* is one result of extreme torsion of the pedicle of an ovarian tumour; but the same accident may occur to a small ovary adherent to omentum. Hence, when after removing an ovarian tumour the surgeon finds nothing but a stump on the opposite side, he should carefully search for the missing ovary higher up in the abdomen. Arrested descent of the ovary will be discussed under "Hernia of Ovary."

ATROPHY AND HYPERTROPHY OF OVARY.—*Atrophy* before the menopause is the rule in wasting diseases. It must not be confounded with imperfect development of the ovary described above under the heading "Rudimentary Ovaries," nor with cirrhotic contraction from inflammation. Both these conditions are usually associated with pain. It is doubtful whether pure uncomplicated atrophy is ever painful. *Hypertrophy* pure and simple is a doubtful condition, not to be confounded with œdema and hydrops folliculi.

HERNIA OF OVARY AND ALLIED AFFECTIONS must here be considered in relation to the ovary alone. As varieties of rupture they have been described under "Hernia." *Arrested descent* has been observed by Martin in three ovariectomies. The normal ovary was detected high above the pelvic brim, against the side of the lumbar vertebræ, immediately under the kidney. *Prolapse* into Douglas's pouch, when not due to inflammatory swelling or adhesion, is akin to hernia. In Ward Cousins' case, in a lunatic aged fifty, there was bad prolapse of the uterus and rectum. Rupture of the vagina occurred during violent straining, and one ovary and tube protruded through the wound. They were successfully removed.

*Hermaphroditism in Congenital Inguinal Hernia*.—An ovary-like structure in such a hernia has repeatedly proved to be a testicle (Chambers, Martin, G. R. Turner, F. Neugebauer, thirty-three cases). In the most recent case (Turner's) a movable, oval, irreducible swelling was removed from the left inguinal region, from a "girl" aged fourteen, ruptured from her birth. It proved to be a testicle; then atresia vaginæ and absence of any sign of a uterus were detected. Hence in any case of congenital inguinal hernia the external and internal genitals should be explored carefully.

*The ovary in a hernial sac*, "ovarian hernia," with no doubt about the nature of the ovary, is frequent. The hernia is usually inguinal (excluding all doubtful cases), but may be femoral, umbilical (Solvieff), obturator, or even ischiatic or gluteal (Waldeyer, Routier). The homologies of the normal descent of the testis and the abnormal descent of the ovary are considered by Lockwood, Klaatsch, and W. M'A. Eccles. Wiart and others believe that hernia of the tube precedes that of the ovary (see "Fallopian Tubes, Diseases of"); but Kossmann has collected six cases of inguinal ovarian hernia, where the tube had not entered the sac. Though congenital inguinal hernia of a genuine ovary has never been authenticated, acquired hernia not rarely develops a few months after birth. It is perfectly reducible at first, but speedily becomes irreducible. Several successful operations have been performed on infants. The ovary was irreducible in Pinkerton's case (child sixteen months), cystic in Beckett's (eleven weeks, the youngest case), and strangulated in Pollard's (three months), and Hooper May's (seven months). But the ovary was not examined in any of these cases, and it must be remembered that a fimbriated tube has been



found associated with a genital gland which the microscope proved to be a testicle. In the adult a well-developed herniated ovary swells and becomes tender at the period. Conception may occur in a woman, subject for years to double inguinal hernia (Beigel), and Widerstein found an ovary with a gravid tube in a left inguinal hernia (see Kossman).

**HYPERÆMIA, INTERSTITIAL HÆMORRHAGE, HÆMATOMA, APOPLEXY.**—In febrile diseases hyperæmia of the ovary is often detected after death. Diffuse hæmorrhage is a common result, the stroma of the ovary being more or less soaked in blood, a change marked in Asiatic cholera, causing a choleraic pseudocatamenial flow. These hæmorrhages are also seen in cases of typhoid fever and less frequently in the exanthemata, in bad burns, and phosphorus poisoning, and from purely mechanical causes, as twisting of the pedicle (Erfurth, Martin). Hæmatoma is not rare in follicular hypertrophy, to be described under chronic oöphoritis. As the clot is found decolorised in some of the follicles, and more or less recent in others, the process must be chronic. The author has described a case of acute apoplexy of the ovary attended with violent pain and swelling of the organ, which was removed five months later, and found to contain a large decolorised clot communicating with a hypertrophied corpus luteum. Rupture of the ovary in acute apoplexy and rapid death from hæmorrhage has been reported, but not recently, and Bland-Sutton reasonably suspects that the ruptured structure was a gravid tube.

**BLOOD CONCRETIONS IN THE OVARY** are uncommon, and do not seem to be the result of inflammation nor the usual consequence of hæmatoma or apoplexy of the ovary. Most likely, however, they are to be traced to the latter condition. Hector Mackenzie and the author have each described a case, both of which occurred in patients with uterine myoma. In the author's specimen, preserved in the museum of the Royal College of Surgeons of England, the concretions lay in a cyst full of blood an inch and a half in diameter in the right ovary. Bland-Sutton found over a hundred similar concretions in an ovarian cyst full of blood through twisting of the pedicle.

**INFLAMMATION OF OVARY: OÖPHORITIS.**—The ovary is often the seat of inflammation. In such a case the tube and adjacent peritoneum are nearly always involved. Hence oöphoritis is inextricably mixed up with perimetritis and salpingitis, which must be studied and considered in association with the former disease. But an inflamed ovary is a distinct entity, often detected on palpation and often exposed at operation. It exhibits the characteristic appearances of inflammation known to the pathologist.

*Causes.*—Living authorities reject the old idea that chills cause oöphoritis. A pure chill, from sitting in damp boots for instance, may assuredly aggravate a chronic attack. No doubt this disease is, as a rule, due to sepsis, especially puerperal, to gonorrhœa, or to tubercle.

*Classification.*—Excluding tubercular disease, oöphoritis is most conveniently classified after Martin and Orthmann's principles.

*Acute oöphoritis* usually consists in free small-celled infiltration into the stroma of the ovary ("interstitial" form), but the follicles may be involved ("general" form). This disease tends to subside, to end in suppuration, or, most frequently, if neglected, to pass into the chronic form. When symptoms of perimetritis are marked the *diagnosis* of acute inflammation of the ovary is often easy. The ovary is distinctly swollen and very tender. The longer the inflammatory symptoms have lasted, and the more the ovary has become adherent to adjacent structures, the harder will diagnosis become. As to *treatment*, a recently inflamed ovary, still movable, should never be removed. Under the usual treatment



suitable for acute perimetritis, it will soon recover provided that adjacent structures be not gravely involved.

*Chronic oöphoritis* is extremely common and hardly ever uncomplicated. As in the acute form, it may be "interstitial," or the follicles may be involved ("general" form). The chronic inflammatory process is usually destructive to the follicles, whilst great hypertrophy of the stroma occurs, not to be mistaken for fibroma. The vessels in the hilum become dilated. Instead of this false "fibroma" developing, the stroma may atrophy, whilst the follicles, thickened and unable to rupture, become dropsical. This is the condition known as "follicular hypertrophy" or "small cystic degeneration." A few proportionately very big cysts fill a small ovary. The interest of this second form of chronic oöphoritis lies in the difficulty of diagnosing it from early cystoma proper. There is a third form of chronic oöphoritis where the stroma shrinks and becomes hard, whilst the follicles disappear. This variety is often termed "cirrhosis of the ovary."

*Abscess of the ovary* may occur after acute oöphoritis, or set in during chronic inflammation. In sepsis after childbirth diffused or interstitial suppuration in the stroma sometimes takes place. The follicles, and even the corpora lutea may become the seat of abscess. In chronic oöphoritis, the abscesses seem often due to secondary infection of the hypertrophied follicles, a change homologous to suppuration of a true cyst and to suppuration of a hydrosalpinx. *Corpus luteum abscess* may develop in the same manner; but in the case of a corpus luteum, where mechanical rupture has occurred, it is easier to understand how infection follows if there be close adhesions to the bowel or to a pyosalpinx. Fraenkel reminds us that gonorrhœal infection of the tube makes infection of the follicle opening for ovulation quite possible.

TUBO-OVARIAN CYSTS are described under "Fallopian Tubes, Diseases of." One variety must be noticed here, that which results from the union of an adherent dilated tube to an ovary undergoing follicular hypertrophy. This form, which is of purely inflammatory origin, was distinguished independently by Schramm and Nielsen and by the present author.

SYPHILIS.—Recent research has failed to prove that this disease produces any special lesion in the ovary. Records of gumma of the ovary, often quoted, are scanty and untrustworthy. A true *syphilitic oöphoritis* has never been satisfactorily distinguished. Women infected with syphilis are liable to suffer from oöphoritis due to other causes than syphilitic infection.

Though the ovary has been found more than once infected with the parasite of *actinomycosis*, it seems never to be the primary seat of that disease. Precisely the same may be said of the ovary in *leprosy*, according to Babes' report, read in 1897 at Berlin before the International Leprosy Congress.

TUBERCLE OF THE OVARY.—This organ does not offer a favourable nidus for tubercular infection, and hence in comparison with the tubes, or even the uterus, it is rarely infected in common tubercular diseases like phthisis. Dr. P. Dymock Turner found undoubted tubercular lesions in the internal genitals, in five out of twenty-seven consecutive necropsies on women who had died of phthisis in the Brompton Hospital. The ovary was involved in one case only, and in that instance the uterus and tubes were also tubercular. Orthmann denies that *primary tubercle of the ovary* has ever been detected. In von Franqué's case the ovary was removed through the vagina and the patient recovered; the primary seat of disease might have been in another organ. *Secondary tubercle of the ovary* may be



commoner than is generally suspected. In nearly 16 per cent of recorded cases of genital tuberculosis the ovary was reported to be infected on the strength of naked-eye appearances, but recent reliable statistics raise the percentage to 34 (Orthmann). The peritoneum or Fallopian tube is almost exclusively the source of infection.

Tubercle of the ovary may manifest itself as miliary or caseous deposit in the stroma or on the surface of the organ; but the most important form is the *tubercular abscess of the ovary* containing characteristic pus. As in tubercle elsewhere bacteriological evidence may be easy or difficult to obtain. *Tubercular tubo-ovarian abscess* has been reported, but the sources of fallacy noted in "Fallopian Tubes, Diseases of," should be remembered; in other words, a large tubercular pyosalpinx may have been taken for a tubo-ovarian cyst.

*Symptoms.*—Pelvic pain and swelling and evidence of suppuration have been recorded in cases of this disease. *Diagnosis* is hardly possible, for when these symptoms are observed tubercular pyosalpinx is much more probable. *Local treatment* is not satisfactory. It has been noticed that after simple incision through the vagina the discharge has been very obstinate, leaving a fistulous track. Abdominal section may cause rapid dissemination of tubercle. Therapeutics are safer than surgery in tubercular ovarian disease.

TUMOURS OF THE OVARY are *cystic* or *solid*. The former are more specially associated with that organ, and their pathology is somewhat complicated. Hence, to avoid confusion, their histogenesis will be considered before the characters of the fully-developed cyst, as known to the surgeon, are described.

*Histogenesis of Common Type of Ovarian Cyst.*—The "glandular cystoma ovarii," the "cystadenoma pseudomucinosum," or common ovarian cyst, undoubtedly arises from structures in the oöphoron or cortex of the ovary, and bears a close resemblance to an adenoma in other parts of the body. A good deal is known about the cellular elements on the surface of the foetal ovary, and about the histology of the Graafian follicle from foetal life to old age. Nevertheless, the precise histogenesis of this well-known morbid growth has not been determined to the satisfaction of all pathologists. The histology of the primary, the growing, and the ripe Graafian follicle is described in the article on GENERATION, FEMALE ORGANS OF. The epithelial lining never assumes the character of the rows of large columnar cells which line the inner wall of an ovarian cyst. Some pathologists still trace this cyst to the superficial epithelium of the ovary, normally cylindrical, a strong point of evidence in favour of their theory as the epithelium of the cyst is of the same type. The superficial columnar epithelium, according to their theory, becomes invaginated into the ovarian stroma; but there is some difference of opinion as to how this invagination occurs. Some, including certain very recent observers, believe that portions of the superficial epithelium become buried in the cortex by inflammatory or other accidental changes (Nagel, Gusserow, Opitz). Indeed, Opitz traces uterine and tubal myoma as well as ovarian cystic disease to inflammatory changes. Others look on the involution as a teratological condition. Some, maintaining that the involuted structures normally develop into some or all of the elements of an ovum and its follicles, believe that through some morbid influence some of those structures develop into a cyst. This theory comes near to the doctrine that the glandular cyst is developed from a previously normal follicle, the change having nothing to do with any involution. This doctrine is plausible, but objectors immediately point out the difference in the



epithelium in the follicle and in the tumour. Yet amongst current authorities Hofmeier had detected and figured epithelial involutions from follicles forcing their way into the stroma, whilst Pfannenstiel has recorded and sketched the remains of an ovum in a minute cyst lined with a perfect layer of columnar epithelium, and adjacent to undoubted glandular cystic structures. We must not deny that the cubical epithelium of the follicle can, under any circumstances, alter its character. Pozzi and Beaussenat believe that ovarian cysts may develop from both the sources above mentioned. They certainly seem to prove, on the evidence of sections from one case, that at least cancer of the ovary may develop from the epithelium of the Graafian follicles. In the further study of this subject care must be taken about conclusions drawn from appearances in sections of a small ovary, the fellow to a glandular ovarian cyst. Such appearances may erroneously be taken to indicate incipient cystic disease.

*Histogenesis of the Papillomatous Cyst.*—The “cystoma ovarii papillare,” or “cystadenoma serosum papillare,” is certainly different from the commoner or glandular cyst. Whitridge Williams claims to have detected papillomatous growths sprouting from the interior of a well-developed Graafian follicle. But Wendeler and others threw some doubt on Williams’ theory, based on five specimens, that papilloma arises from the Graafian follicles. It is not clear how many of the ovaries examined by that observer were really about to become papillomatous cysts. The older theory that papillomata arise from relics of the mesonephron, portions of the Wolffian ducts which penetrate the paroöphoron, or even enter the oöphoron, was strongly maintained by Coblenz. Bland-Sutton and the author have supported it on naked-eye evidence. This kind of cyst tends to burrow between the layers of the broad ligament; now the paroöphoron lies in the folds of the broad ligament. Pure papilloma of the broad ligament, with the adjacent ovary unaffected, is not rarely seen, and would appear to arise from the parovarium or mesonephron. On the other hand, cystic papilloma may form a well-pedunculated tumour like a glandular cyst, or papillomatous and glandular cysts may be blended in one pedunculated ovarian tumour, as though relics of the mesonephron which normally exist in the oöphoron had given rise to tumours of this class. Kossmann has recently traced papillomatous tumours to Müller’s duct. He has certainly succeeded in showing that minute “accessory Fallopian tubes” bearing fimbriated epithelium are often scattered over the surface of the broad ligament and ovary. The ovarian fimbria, lined with similar epithelium, sends detached processes on to and even into the ovary. These scattered elements tend, Kossmann maintains, to become encysted. Still the parovarian or mesonephric origin of papillomatous cysts seems based on stronger evidence.

*Histogenesis of Dermoid Cysts.*—The well-known tumour which contains epidermic structures, bone, nerve, etc., develops in the oöphoron. We know that it does so; our ignorance why it does so must not be covered by cunningly devised phrases like “foetal inclusion” or “parthenogenesis.” One of the very latest writers traces dermoids to “atypic, parthenogenetic ova.” There seems little doubt that they arise in Graafian follicles. Pregnancy in a follicle, we must remember, is almost if not quite unknown. Even van Tussenbroek’s case of alleged early ovarian gestation may be akin to an incipient ovarian dermoid. It has been hinted by Bland-Sutton and others that dermoids and common glandular cysts are closely related, the latter being tumours with the power of developing mucous glands, the former tumours that can develop a far greater variety of structures.

**SIMPLE CYSTS: DROPSY OF THE FOLLICLES.**—This affection is apparently



nothing more than an extreme form of the "follicular hypertrophy" already described as a variety of chronic oöphoritis. The ovary may become as large as a hen's egg; the dilated follicle sometimes bears a layer of cubical epithelium on its inner wall; two or three such cysts are not rarely found ("oligocystic tumour"). It is not always easy to distinguish these cysts, direct results of inflammation, from an incipient cystoma. This fact accounts for the differences of opinion amongst authorities on the nature of small cysts. *Corpus luteum cysts* are probably of the same origin; they may exceed two inches in diameter.

THE COMMON OVARIAN CYST.—This tumour is also termed "cystadenoma pseudomucinosum," or glandular ovarian cyst. Its outer wall is at first silvery-white, with moderately large vessels, but when extensively riddled with minute cysts it appears greenish and gelatinous. Other appearances, such as a dull surface, coloured patches, etc., are due to inflammation, hæmorrhage, and degeneration. This cyst is always multilocular. Sometimes one loculus predominates, the others may even be but microscopical; in other cases, especially when there is much glandular substance, there are several loculi of much the same size. When the loculi are partly separate externally the tumour appears like a pile of cannon balls. In a predominating cavity it is the rule to find little or no solid matter, and the internal lining membrane is usually divested of epithelium, owing to maceration, but this variety should be ranked with glandular cysts, there being no true line of demarcation between them. In the opposite type large masses of glandular growth project into the cavities of the larger loculi, and almost fill up the smaller loculi, whilst there are usually large areas where the solid predominates over the cystic element, and where the cavities are only indicated on section by specks of dead white mucoid material, the secretion from the dense masses of glandular matter. The walls of the loculi are firmest where there is least infiltration with minute glandular cysts, and are most vascular at such points. Hence there is sometimes much loss of blood when the surgeon breaks down the loculi to facilitate extraction.

The fluid contents consist in the large loculi of a glairy albuminous fluid. This is typically greyish green, and seldom approaches the yellow tint of ascitic effusion. But ovarian fluid is often dark brown or otherwise discoloured by hæmorrhage, and often mixed with semi-opaque dead white lumps, the product of active secretion from glandular growths. In the smaller loculi these lumps may constitute the sole contents, when very dense they appear bright white. In some cysts the contents are entirely colloid in consistence, and devoid of the least fluidity. Amidst the loculi dermoid and papillomatous or even malignant growths may be found.

General pathologists do not hesitate to rank this familiar tumour as an adenoma (Sims Woodhead, D. J. Hamilton). The lining membrane of the predominating cystic cavity bears either flattened cubical epithelium or is entirely divested of any epithelial covering. The minute cysts are lined with very large columnar epithelium, including numerous chalice cells. This adenomatous tissue may implant itself on the peritoneum, a change as a rule clinically malignant.

The pedicle is, in the majority of cases of common ovarian cyst, anatomically normal, though always hypertrophied and often exceedingly broad. Only a small proportion burrow into the mesosalpinx, very few into the mesometrium. This subject will be further discussed under the paragraphs on "The Pedicle" and "Capsules, True and False." In about one-third of all cases the common ovarian cyst is bilateral (Stoecklin).

THE PAPILLOMATOUS OVARIAN CYST must be classed with a non-cystic



ovarian tumour, *free papilloma of the ovary*, where masses of papillary growth spring from the surface of the ovary. These free growths set up ascites, and are nearly always bilateral. The rare but conspicuous racemose "Rokitansky's tumour," which resembles a large hydatidiform mole, and has been figured by Lawson Tait, is considered by Stoecklin (Martin, *loc. cit.* p. 524) to represent œdema or myxomatous degeneration of free papillomata. The pseudocysts are certainly not ova or follicles. The true *papillomatous cyst* is frequent; it is often bilateral. Often the smaller growth is but little larger than a normal ovary, and hence liable to be overlooked. Pfannenstiel maintains, on statistical evidence, that such an ovary should always be sacrificed, and the author's experience induces him to support that principle.

The outer wall of a papillomatous tumour is usually thin; contrasted with the surface of a glandular cyst it is dull in colour. As a rule the tumour consists of a few cavities of fairly equal size. From its inner wall spring the characteristic papillomata, some appearing as mere rough elevations, some as large warts, others as luxuriant cauliflower masses, very vascular and sometimes gritty through psammomatous changes. These growths frequently burst through the outer wall, which often bears similar growths extra-cystic from their origin. The fluid contents are as clear as water, devoid of albumin, and rich in chloride of sodium. The papillary growths consist of columnar, usually ciliated, cells of moderate size, mounted on a very scanty framework of connective tissue, with large vessels to each papilla.

A minority of papillomatous cysts, according to the author's experience, bear a normal pedicle. As a rule they are sessile, and not rarely burrow into the mesometrium as well as into the mesosalpinx. Implantation of papillomatous growths on the peritoneum is common, it may prove clinically malignant within a few months; yet the growths may wither completely after removal of the tumour.

DERMOID CYST (or "cystic ovarian embryoma") is commoner in the ovary than in any other part. It arises from the ovary proper, and hence is nearly always pedunculated. The pedicle is particularly liable to torsion, ending sometimes in complete detachment. About 15 per cent of all ovarian cysts are dermoid, and a large proportion of dermoids are bilateral; in infants and under puberty, ovarian cysts are nearly always dermoid. The outer wall of a dermoid is usually dull white and often discoloured. Its fluid contents present great variety, but are always more or less greasy. The grease may be clear liquid fat. Sometimes the fat forms itself into a large collection of white pills; when mixed with sebaceous material it appears like yellow pomatum. Free hair, loose or in dense, felted balls, is very frequent. From the walls spring, in most dermoids, amorphous masses of bone often bearing teeth, or teeth may be found without bone. The inner lining of the cyst is skin bearing sebaceous and sweat-glands. Deeper under the surface nerve and muscle and many other structures may be found.

*Teratoma of the ovary*, "solid ovarian embryoma" (Wilms), is an essentially solid growth. It arises in a limited area of the ovary, the remainder of which is often detected in its capsule. Structures derived from all three layers of the blastoderm are found arranged without any regularity; in some cases they simulate an amorphous acardiac foetus. It grows rapidly and is highly malignant, clinically speaking, though it does not undergo cancerous or sarcomatous degeneration. About fifteen genuine cases have been described.

*Malignancy of Dermoids and Teratomata.*—The frequency of sar-



comatous or carcinomatous degeneration has been exaggerated, soft connective tissue in a dermoid being often falsely reported as sarcoma (Bland-Sutton). M. Sanger (Martin, *loc. cit.* p. 677) finds that true malignant degeneration only occurs when the dermoid has grown large. He reports and figures a case of carcinomatous degeneration of a dermoid (p. 696), and also an instance of cancerous infection of a dermoid from primary disease in the rectum (p. 698). But "epithelial infection" (Bland-Sutton) or "peritoneal implantation" (Fraenkel, Kolaczek), a clinically malignant change where dermoid masses are disseminated over the peritoneum, is frequently observed. Teratoma invariably and rapidly spreads beyond the ovary in this manner.

FIBROMA OF THE OVARY is relatively frequent about the menopause and in girlhood. When soft it is often taken to be a sarcoma. A hard and painless tumour in the hypogastrium, moving separately from the uterus, is most probably an ovarian fibroma, especially in a young subject free from cachexia and amenorrhoea. It causes remarkably little pain, is seldom adherent, and is often associated with a moderate amount of ascites. In one case the author found a large ovarian fibroma firmly impacted in the pelvis. The pedicle is usually short and bears large vessels, so that much care is needed in securing it during ovariectomy. It is sometimes twisted without producing any severe symptoms such as are seen in twisted dermoid. Ovariectomy is the only treatment, and is very successful.

MYOMA OF THE OVARY is usually small; the author removed a small tumour of this kind where the uterus was free from "fibroids." On section the fibres were seen to be arranged in spherical nodules, like balls of worsted. About a dozen authentic cases have been recorded.

ENCHONDROMA.—Areas of cartilage, sometimes ossifying, have been detected between the fibres of an ovarian fibroma. One such tumour weighed over 14 lbs. (Buet, *Union Medica.*, No. 4, 1900).

SARCOMA AND CARCINOMA.—Through careful research, conducted on a large scale in Germany and Austria, pathologists have found that primary sarcoma is rarer in the ovary than primary carcinoma. In 307 cases of malignant tumour in the practice of Olshausen (Kratzenstein's returns). Leopold, Martin, and Cohn, 69, or only a little over 22 per cent, were sarcomata. These figures and calculations, however, can never be thoroughly verified. Martin insists that endothelioma must be ranked under sarcoma, whilst Bland-Sutton feels certain that many tumours of the ovary described as primary carcinomata prove on careful examination to be sarcomata.

*Sarcoma* may be of the spindle-celled variety, then it is relatively firm, often blended with fibrous tissue, and of somewhat slow growth. This is the kind of tumour so difficult to distinguish from fibroma of the ovary. The round-celled varieties form much softer and more malignant growths. There is also an angiosarcoma of the ovary, and an interesting variety, *endothelioma*, developed from the endothelium of the blood-vessels or, in a sub-variety, from the endothelium of the lymphatics (Eckardt and Herz). An endothelioma may be firm (Pick), soft (Amann), or cystic (Olshausen). Cystic changes, bleeding and well-known retrograde conditions, are frequent in sarcoma. Often sarcoma of the ovary is bilateral, more likely from metastasis from one ovary to its fellow than from simultaneous malignant infection.

Sarcoma often forms a very large tumour which may be regular in outline to the last. Amenorrhoea and ascites tend to appear early. Metastatic deposits usually develop later than in cancer; but cystic



sarcoma, especially when developing in a common glandular cyst, is clinically very malignant. The omentum and intestine are specially exposed to infection.

*Carcinoma* appears to be the commoner form of malignant disease of the ovary. It may develop directly from the epithelial elements in that organ or from the common glandular cyst (malignant adenoma). Papilloma occasionally, but rarely, undergoes cancerous degeneration not to be confounded with implantation of papillomatous growth on viscera and peritoneum. Cancer tissue is often firm at first, but is apt to alter in consistence through the free development of cystic cavities, which is part of the cancerous process independent of any pre-existing innocent adenomatous cyst. The cancerous ovary appears as a tumour, usually irregular in shape and consistence. In over 50 per cent of all cases this disease is bilateral, but as in the case of sarcoma simultaneous infection is doubtful (Martin).

Cancer of the ovary is, as compared to uterine and mammary cancer, rather slow in growth. The pedicle isolates the tumour for a while. But when once the peritoneum becomes infected the disease advances rapidly and extends to the lumbar glands. Ascites and often amenorrhœa set in early as in sarcoma.

*Age and Malignant Ovarian Tumours.*—Carcinoma, as well as sarcoma, seems distinctly most frequent in young girls a little over puberty, and at the menopause, but both diseases have been seen in infants and old women.

*Ovariectomy and Malignant Disease.*—In the early stages the operation is easy, for malignant tumours are usually well pedunculated. When the tumour forms a fixed mass no attempt must be made to remove it. In the middle stages ovariectomy is practicable, but the mortality is high; nevertheless, it should be undertaken, for though recurrence is usually rapid, especially in cancer (Sänger, Pfannenstiel), about 40 per cent of the patients live for over a year free from recurrence (Kratzenstein).

**THE PEDICLE.**—A pedunculated ovarian tumour lies in the peritoneal cavity like a normal ovary. Its surface is not invested by any part of the peritoneum, not even by that portion which forms the broad ligament. It is connected with the uterus by the Fallopian tube, by its own "ovarian ligament," and by the mesosalpinx between the tube and the ligament, and is united to the peritoneum of the brim of the pelvis by the infundibulopelvic ligament which contains the ovarian vessels and joins the broad ligament. These connections together make up the "pedicle." The anatomy of the parts concerned (see "Broad Ligament, Diseases of") should be thoroughly understood by the surgeon. The big ovarian vessels lie in the outer border; and close to the inner border lies the anastomosis of a large branch from the ovarian, with another from the uterine artery. The Fallopian tube and mesosalpinx are nearly always hypertrophied in the pedicle of a common multilocular cyst, indeed in most cystic tumours; on the other hand, in many cases of solid and dermoid tumours no such overgrowth occurs, even when the tumour is very large.

The operator can always recognise a true pedicle by the position of the tube and mesosalpinx, which are seen lying on the tumour, attached to, but distinct from it.

The great majority of simple, multilocular and adenomatous cysts, of dermoids, and of solid or semi-solid tumours, together with a minority of papillomatous cysts, are pedunculated.

**CAPSULES, FALSE AND TRUE.**—In most cases a *true capsule*, that is to say, an investment of peritoneum which the tumour makes for itself, consists



of the mesosalpinx alone. The surgical anatomy of the broad ligament is demonstrated in the article "Broad Ligament, Diseases of." A knowledge of the anatomy of capsules is of high importance to the surgeon.

Fig. 1 is a diagram of the broad ligament seen in vertical section midway between the uterus and the pelvic wall. Fig. 2 shows how a simple or papillomatous cyst of broad ligament origin (such as the common "parovarian" tumour) is encapsuled in the mesosalpinx. In Fig. 3 the mesosalpinx forms a capsule for a tumour (usually papillomatous) developed in the hilum or parenchyma of the ovary itself. Tumours with the mesosalpinx as a capsule can be recognised by the Fallopian tube which runs, greatly stretched, over the surface of the capsule.

The surgical anatomy of the broad ligament

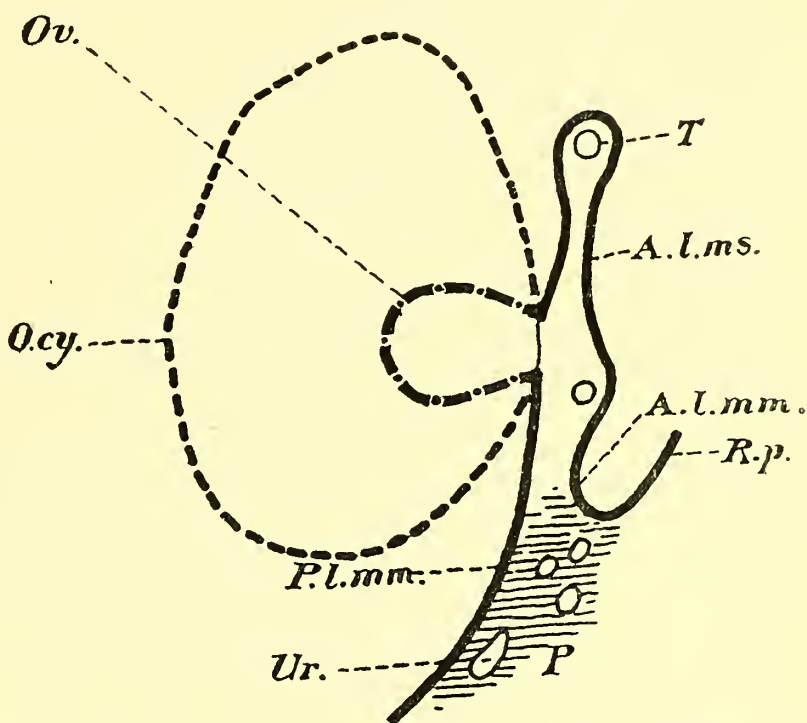


FIG. 1.—Normal relations of ovary to broad ligament and tube. *O.cy.* indicates position of an ordinary ovarian cyst, which does not burrow. The round ligament is indicated between *A.l.ms.* and *A.l.mm.*, but omitted in the other drawings, to avoid confusion. The lettering is fully explained under Fig. 4.

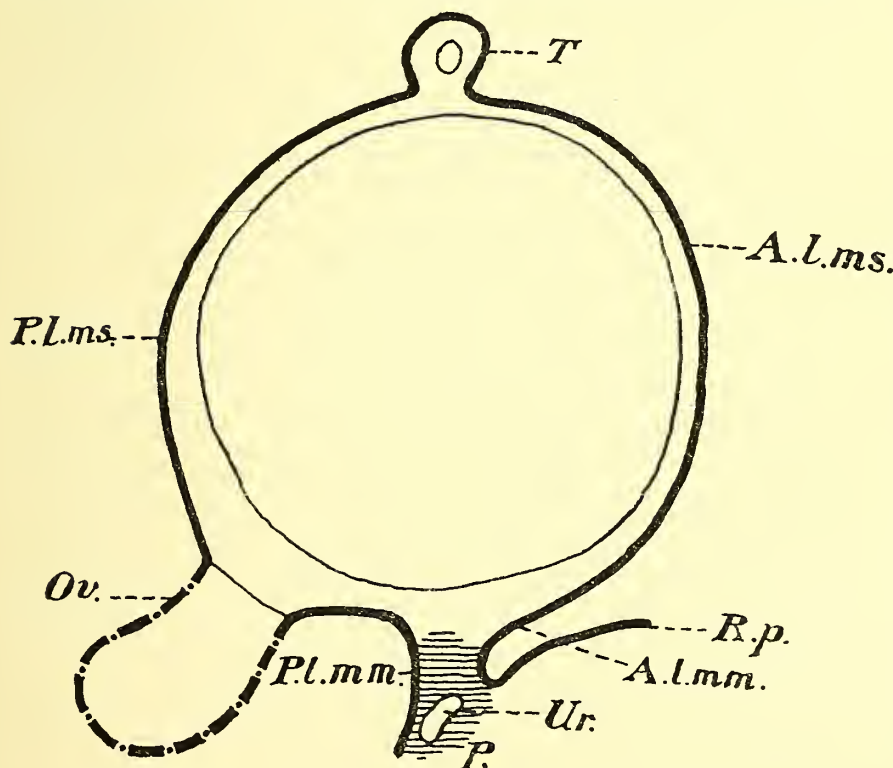


FIG. 2.—Common "parovarian" or broad ligament cyst in mesosalpinx.

as it rises, displaces the entire anterior layer of the broad ligament and its reflexion (*R.p.*) on to the abdominal wall. Hence the tumour is devoid of peritoneum over the greater part of its anterior aspect. The same relations are seen in "anterior tubo-ligamentary pregnancy" (Hart and Barbour, Taylor). Sometimes the tumour displaces the posterior layer of mesometrium so as to become entirely retro-peritoneal; this is occasionally seen in the case

of the capsule. In Fig. 4 an ovarian, and in Fig. 5 a pure broad ligament cyst are represented encapsuled not only in the mesosalpinx, but also in the mesometrium, that portion of the broad ligament which lies below the level of the ovary. In such cases the base of the tumour lies very low down in the pelvis below the normal level of the cervix uteri; the uterus is sometimes displaced upwards, lying flattened in the surface of the tumour. The tumour,

of an ovarian cyst, and when the cyst is a tubal gestation sac it constitutes

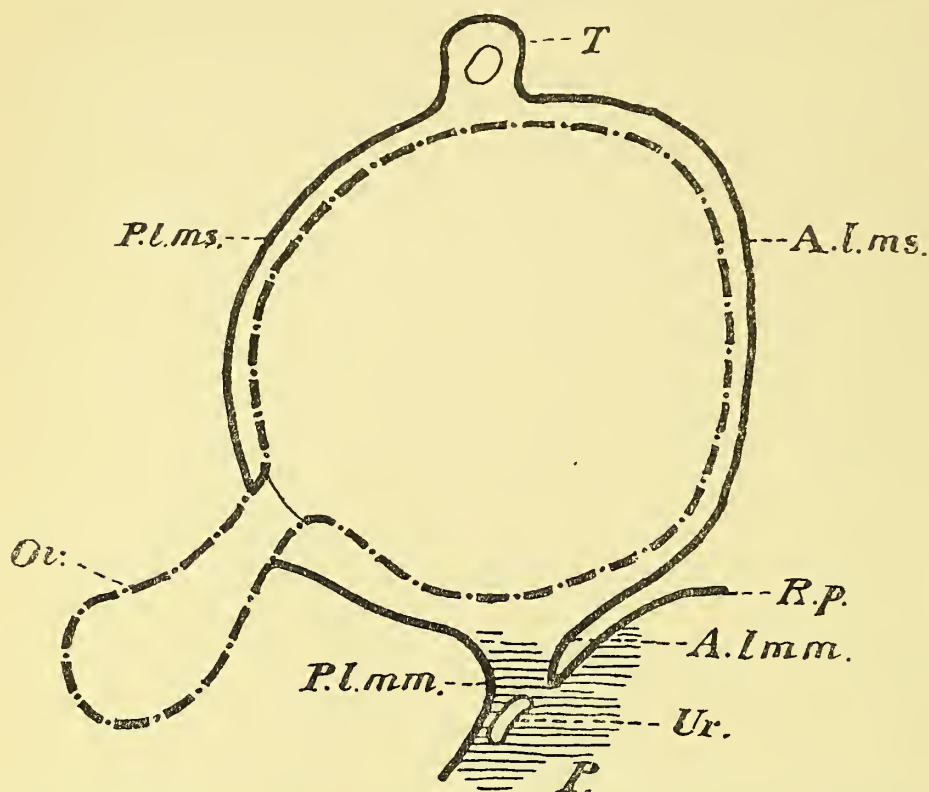


FIG. 3.—Cyst of ovarian origin burrowing in mesosalpinx (usually papillomatous).

This is the usual form of encapsulation in fibroids of the broad ligament; in such a case not only the mesosalpinx and tube, but the ovary as well, lie on the tumour. The operator must remember that all tumours burrowing into the mesometrium come into close relation with the ureter and also with the rectum.

*False Capsule.*—Pawlik distinguishes a “pseudo-intraligamentary ovarian tumour.” In this condition the tumour appears as though encapsuled between the folds of the broad ligament, when in reality the tube and mesosalpinx are plastered down on to the surface of the tumour by firm adhesions, and the lower parts of the tumour are strongly adherent to Douglas’s pouch (Fig. 7).

**COMPLICATIONS.**—The most remarkable complication associated with ovarian tumour is twisting of the pedicle, though not absolutely peculiar to this form of growth. It will therefore be specially considered.

*Ascites* occasionally develops in simple cases of common ovarian cyst;

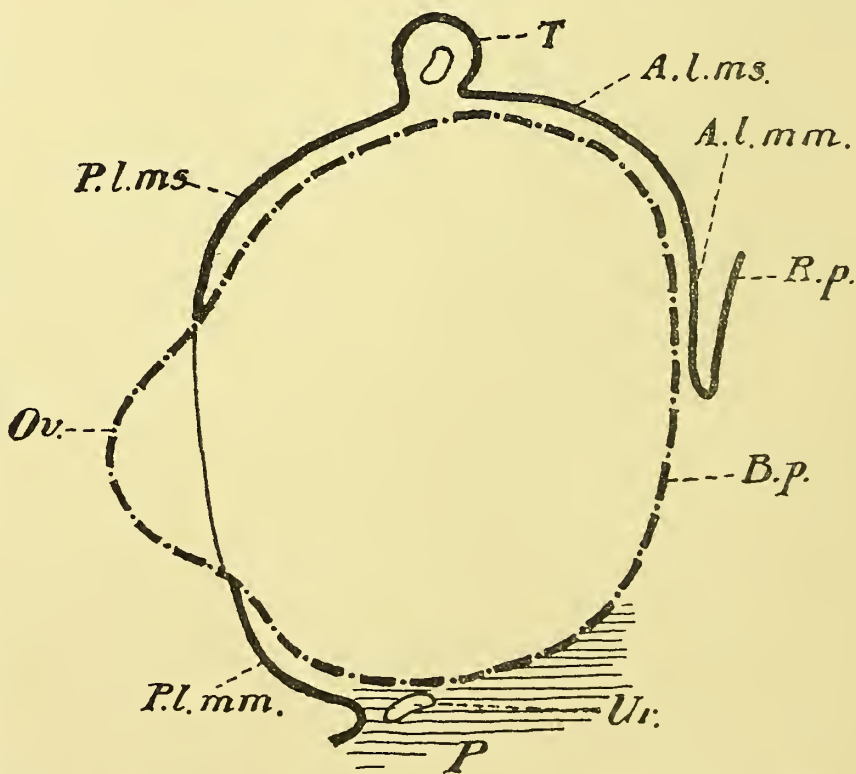


FIG. 4.—Cyst of ovarian origin burrowing both in mesosalpinx and mesometrium. *Ov.*, ovary. *P.*, parametrium or pelvic connective tissue, shaded. *A.l.ms.*, anterior layer of mesosalpinx. *P.l.ms.*, posterior layer. *A.l.mm.*, anterior layer of mesometrium (the part of the broad ligament below the level of the ovary—Waldeyer). *R.p.*, reflexion of peritoneum from mesometrium on to the pelvis and abdominal wall. *B.p.*, part of tumour bare of peritoneum. *Ur.*, ureter.



the author has observed much ascites where the cyst wall was smooth, where no evidence of rupture existed, and where there was no trace of malignant growth or papilloma. In dermoid disease it is, for no evident reason, not common. In freepapilloma and in papillary cysts, where some of the growths have perforated the cyst wall, it is always present, and usually to an extensive degree. It is often present in fibroma, and develops early in all forms of malignant disease. When the patient suffers from a visceral disease

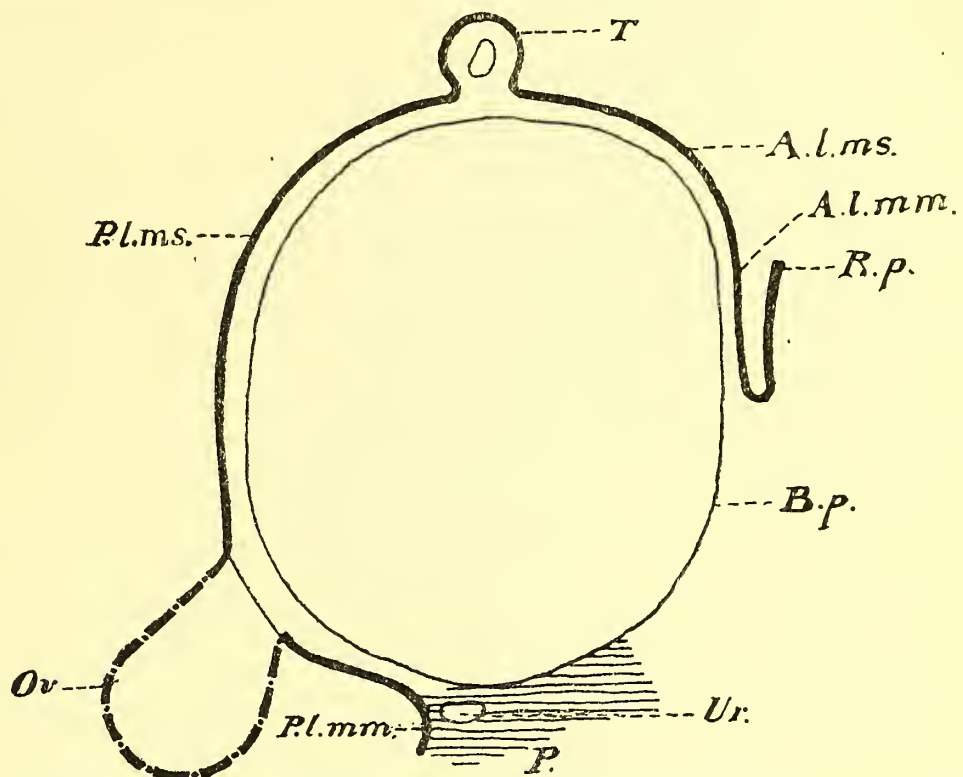


FIG. 5.—Broad ligament cyst, not of ovarian origin, burrowing into mesosalpinx and mesometrium.

which causes ascites, the presence of an ovarian tumour hastens the appearance of that complication for evident reasons.

*Pleural effusion* is, of course, grave when associated with carcinoma or sarcoma, but is not pathognomonic of malignancy.

*Inflammation of the tumour* is an important complication, as it tends to the development of adhesions, and may end in suppuration. The clinical

history is sometimes, but not always clear. There is usually fever and dull pain. These symptoms may be contrasted with the acute localised pain with general good health observed in typical cases of acute torsion of the pedicle. *Suppuration of the tumour* is mostly due to infection from adherent intestine. It is relatively common after labour, and a suppurating tube may infect a tumour. It has been observed to follow typhoid fever.

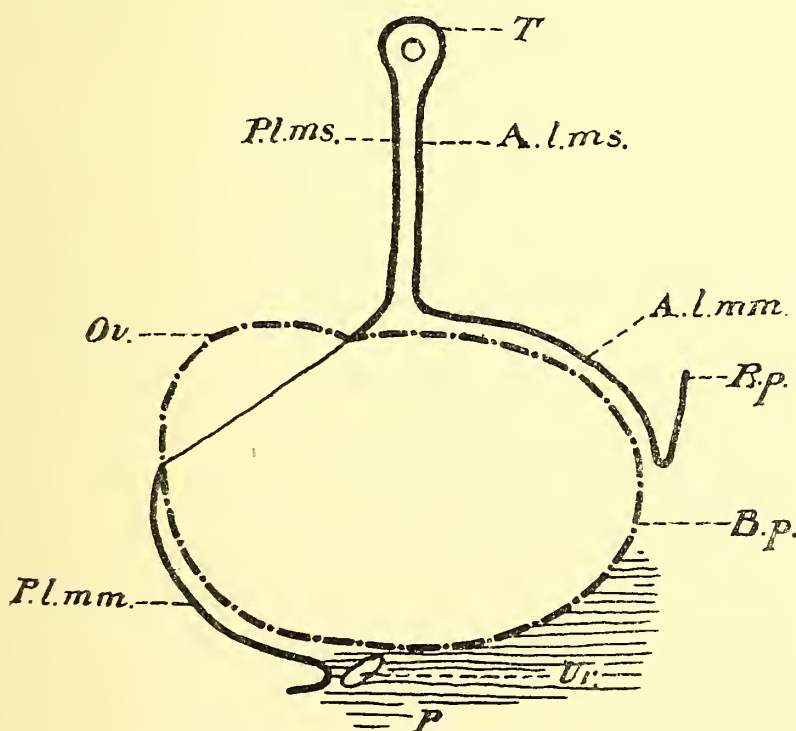


FIG. 6.—Cyst of ovarian origin burrowing into mesometrium, leaving mesosalpinx intact.

usually due to local *peritonitis*, often primary, being caused by the irritation of a tumour itself free from any inflammatory process. Dermoids, seldom associated with ascites, are very apt to set up localised peritonitis, even

But inflammatory symptoms in a case of tumour of the ovary are

when they are small. The same complication always appears when a common ovarian cyst is allowed to grow to a large size. Traumatism, the

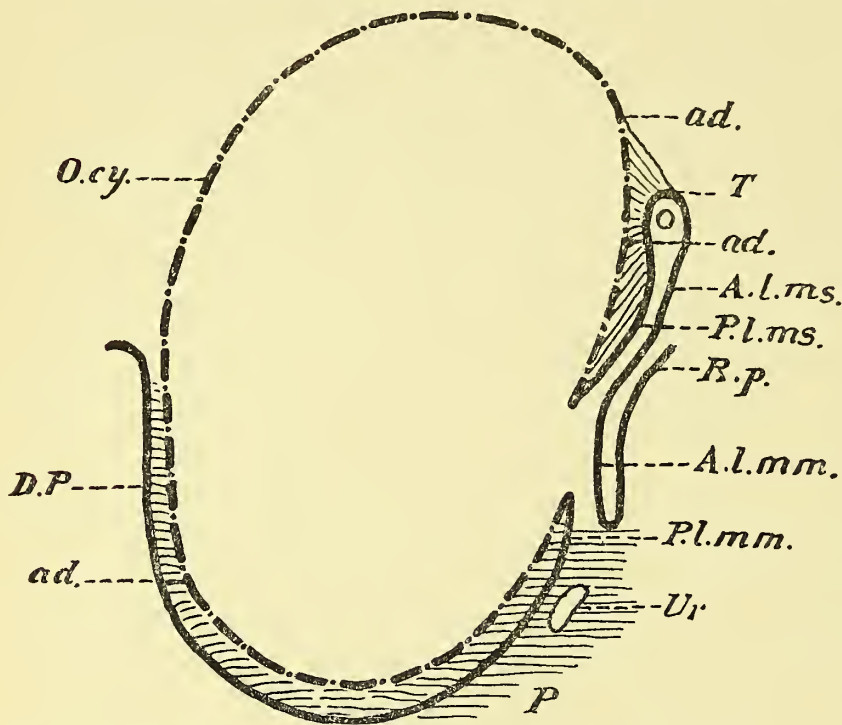


FIG. 7.—Pawlik's "pseudo-intraligamentary ovarian tumour." It is an ovarian cyst which, like *O.cy.* in Fig. 1, does not burrow. *ad.*, adhesions plastering the tube and mesosalpinx and uniting the peritoneum of Douglas's pouch (*D.P.*) to the cyst.

friction of the tumour against the viscera, pregnancy, and many other causes, produce the complication. The *treatment* consists in absolute rest, antiphlogistics, and hot applications for a day or two, and then ovariectomy; but when there is a suspicion of suppuration immediate removal of the tumour is demanded. Great care must be taken when tapping a suppurating tumour during ovariectomy lest any of the contents escape into the peritoneal cavity. The removal of suppurating dermoids strongly adherent constitutes

one of the most dangerous varieties of ovariectomy.

*Adhesions* are the commonest results of inflammation. Parietal adhesions are very common, omental nearly as frequent. In old, neglected, and malignant cases coils of small intestine may adhere strongly. Adhesions to the uterus and bladder are not common, they may be very intimate. The surgeon must be careful to distinguish between adherent large intestine and close relation of large intestine to the tumour when the latter has burrowed under peritoneum. Exact *diagnosis* of adhesions is usually impossible, but presumptive evidence is strong when symptoms of chronic torsion or inflammation have been observed. As for *treatment*, it consists in their careful separation during ovariectomy, a purely surgical question relating to abdominal operations in general. It has been assumed that the wounds which the operator necessarily inflicts during ovariectomy, etc., must inevitably be followed by adhesions. Kelterborn has shown that this idea is a fallacy. A series of experiments, in which pieces of parietal and visceral peritoneum were cauterised or snipped off in animals under aseptic conditions, indicated that the wounds thus inflicted do not tend to contract adhesions. The healthy wound in a ruptured Graafian follicle never adheres. The stump of the ligatured ovarian pedicle bears a wounded surface, but though it may adhere even to intestine, it is often found, in second ovariectomies on the same subject, absolutely free from adhesions.

*Rupture of an ovarian cyst* is not rare. A single loculus often gives way, its outer wall being softened by small cysts in its substance, or by thrombosis of its vessels. Sometimes the same changes may cause a large, practically unilocular cyst to give way. Constitutional disturbance and pain are absent, as a rule, in such cases. The patient notices that her abdomen has suddenly grown smaller. Diuresis commonly follows the rupture, diarrhoea is not rare. In most instances the cyst fills again rather rapidly. The chief danger in rupture of a cyst is dissemination of solid contents.



Even adenoid growths may thus become clinically malignant. Diffusion of colloid matter is very prejudicial to the patient. The diffusion of infected fluid material over the peritoneum after rupture is dangerous, but rare, as where the cyst has become infected there are usually protective adhesions. Lastly, rupture of the cyst from a blow or from hæmorrhage into its interior may cause acute symptoms and even speedy death. The cardinal symptom in all these cases is the disappearance of abdominal swelling. Speedy operation is needed in all cases.

*Intracystic hæmorrhage* is rare, except in association with torsion of the pedicle; in the days of tapping it was more frequent. Occasionally quite a pint of fresh or old blood is found in a loculus. When the cyst bursts from and during hæmorrhage the result may be speedy death. An exploratory incision should always be made in a case where there are symptoms of internal hæmorrhage, with diminution in the size of the abdomen. If a ruptured or bleeding cyst be found, the pedicle should be clamped at once, and ligatured at leisure after all clots and fluid blood have been cleared away.

**TWISTING OF PEDICLE:** AXIAL ROTATION OF OVARIAN TUMOURS is a well-known complication. In its slighter forms the operator may fail to recognise this complication; on the other hand, a large cyst often rotates on its pedicle during emptying and extraction in ovariectomy, so that the surgeon may be under the erroneous impression that the pedicle was twisted before the operation. Lehmann and Martin, taking all sources of error into consideration, make out the percentage of twisted pedicle in all cases of ovarian tumour submitted to operation as between 5 and 7. Authorities are not agreed as to what is precisely meant by torsion from right to left, or *vice versa*, hence much confusion in monographs on this matter. The tumour drags on the ovarian ligament and then rotates. The torsion may be very loose so as to produce no effect on the tumour, or very tight so as to strangulate it at once, or to divide itself gradually so that the tumour is cut off completely from its old attachments. The degree of torsion varies greatly, the pedicle may be twisted one-third of a turn, half a turn, or one, two, or three turns; all these conditions are frequent, and associated with varying degrees of tenseness. As many as twenty-five turns have been observed, but they rarely exceed three. Torsion is observed oftener in small than in large tumours; relaxed abdominal walls, and above all, pregnancy and labour predispose the patient to this complication. In several other kinds of abdominal tumour the pedicle may become twisted. A fibroid may twist the lower part of the uterus itself, a pedunculated fibroid may rotate round its pedicle. Pozzi reports torsion of a tubal gestation sac at the end of the third month. Subbotic, who has removed many spleens for malarial hypertrophy, finds that the pedicle is often twisted. Violent action of the abdominal muscles, falls, etc., may certainly be ranked among the causes. Straining during defæcation caused acute torsion in one case under the author's observation, but it is not certain how far the other factors in defæcation assist in causing torsion. The emptying and filling of the bladder may play a part in some cases. As to causes in the tumour itself, irregular growth, want of uniformity in weight at different parts, and a short narrow pedicle, greatly predispose to torsion. Hence dermoids are specially subject to this complication, as are fibromata and sarcomata of irregular form. In bilateral disease one tumour may twist the other, or, as in a case under the author's care, one tumour may undergo chronic rotation, the other becoming suddenly twisted.

*Symptoms and Effects.*—Acute torsion of the pedicle causes violent



abdominal pain and tenderness with distension and vomiting. The presence of a tumour and the absence of positive symptoms of intestinal obstruction indicate the true nature of the case. The tumour becomes strangulated, deeply livid, with ecchymoses without and hæmorrhage within. These appearances have been mistaken for gangrene, just as the symptoms have led to the suspicion of strangulated hernia, ruptured ectopic gestation sac, etc., and to the consequent discovery of an unsuspected ovarian tumour (Cullingworth). In typical chronic torsion the patient suffers from dull deep pains, and there is more or less arrest of the growth of the tumour without the impairment of health observed in chronic peritonitis. But adhesions always exist in these cases, and may involve further mischief, and diagnosis of chronic torsion is not always possible; menorrhagia is frequent in chronic torsion.

*Treatment.*—An ovarian tumour should be removed, torsion of the pedicle is but one more reason for ovariectomy. In chronic torsion the operator will have little trouble with the atrophied, degenerate pedicle, but he may have to separate intimate adhesions. In acute torsion the pedicle must be carefully handled, and ligatured with special skill, as it is usually œdematous and its veins are dilated.

DETACHMENT OF AN OVARIAN TUMOUR FROM ITS PEDICLE is an occasional result of chronic torsion. The tumour receives its nourishment from the omentum or from dense adhesions to the parietes and viscera. More frequently, when the tumour is thus nourished, the pedicle retains its attachment, but is reduced to a thin cord with obliterated vessels. This condition, as well as complete detachment, is usually associated with dermoids.

SYMPTOMS OF OVARIAN TUMOUR.—As long as such a growth is small, it may give rise to no painful or disagreeable symptoms, whether cystic or solid, pedunculated or sessile, innocent or malignant. It is therefore sometimes accidentally discovered during digital exploration of the pelvis. A small, freely movable oval body, not tender, often in such a case proves to be an ovarian cyst. On the other hand, Davenport and the author have published clinical reports of ovarian tumours simulating inflamed ovaries, loose or fixed in the pelvis. Diagnosis without watching the case for a short time is often impossible. If the pain ceases, but the swelling grows larger, it is very probably an ovarian tumour; if the swelling disappears with the pain that swelling was more probably an inflamed ovary.

A typical ovarian cyst when it has risen above the pelvic brim is usually very characteristic. It forms an oval movable mass, well circumscribed, occupying the hypogastrium, iliac, and umbilical regions, and sometimes reaching to the epigastrium, or even extending into the loins. It fluctuates more or less distinctly, and the pulsations of the aorta are not transmitted to it through the stethoscope. On bimanual palpation it can be felt in front of the uterus, its base just coming into the pelvic brim. Any one, and often several of the above features may, however, be absent. Thus it may be very irregular in outline, it may be bound by adhesions down into one iliac fossa or loin; sometimes intestine gets between its lower limits and the pubes; fluctuation or thrill on percussion may be absent if it be very multilocular. A cyst with a long pedicle sometimes manages to get behind the uterus, which is pushed up above the pubes so as to lie on the surface of the tumour, a condition usually associated with completely sessile cysts. A cyst with a very short pedicle drags on the uterus, so that the two appear to move together as though they were one. Hence the cyst may be taken for a uterine tumour, more especially when, as is frequently



the case, the uterine cavity is elongated. The catamenia remain perfectly normal as a rule, but menorrhagia is sometimes noted, especially after twisting of the pedicle or other complications, whilst amenorrhœa is often observed in young subjects when the ovarian tumour is malignant. Unless it has ruptured and begun to refill, an ovarian cyst is usually tense, often so tense as to feel solid. Flaccidity, with fluctuation, is more usual in parovarian cysts.

Solid ovarian tumours present many of the clinical features of ovarian cysts above described, allowance being made for their solidity, which usually transmits pulsations and involves absence of fluctuation. Fibroma and myoma advance very slowly, much slower than cysts, and are occasionally associated with ascites; malignant ovarian tumours grow more rapidly than cysts, and peritoneal effusion sets in early. Papillomatous tumours grow quickly, and even the most innocent may cause great peritoneal effusion. Dermoids grow slowly, feel doughy to the touch, and are seldom associated with ascites, but are very apt to be complicated by peritonitis and twisted pedicle, causing, or independent of, the peritonitis.

As the tumour grows larger the chance of some of the complications above related taking place becomes greater. The bulk causes pressure symptoms, hence œdema of the legs and in common ovarian cysts late dropsy, once very often observed, now rare because of timely operating. As might be expected, dyspnœa becomes very distressing. Hypertrophy and dilatation of the heart has been detected in many advanced cases. The author, referring to a large series of necropsies which he made between 1877 and 1882, finds that chronic inflammatory kidney disease is extremely common in these advanced and neglected cases now rarely seen. All these complications, together with constant mental worry and physical exhaustion, ultimately cause death. Many cases die within two or three years of the first detection of the tumours, yet some, it must be admitted, used to live twenty or thirty years after repeated tapplings. The author observed one case of this kind that lived for over forty years after innumerable tapplings, refusing operation. She died very suddenly during an attack of dyspnœa, with suppression of urine.

**DIAGNOSIS OF OVARIAN TUMOUR.**—A fair consideration of the above clinical symptoms renders diagnosis of an ovarian cyst, or even of a solid tumour of the ovary, fairly easy; but these symptoms, as well as many complications and exceptions, must be studied, and afterwards carefully borne in mind when any one patient is examined.

*Myoma of Uterus or "Fibroid."*—In this disease the tumour forms part of the uterus, and is solid and elastic; the uterine cavity is elongated; menorrhagia is present in certain varieties. When the cervix is involved diagnosis is not difficult; but a pedunculated subserous myoma, without evidence of any other similar growth in the uterus, and with little enlargement of that organ, cannot always be distinguished from a solid ovarian tumour. Uterine myoma grows slowly. *Cystic myoma* is not easy to distinguish from burrowing semicystic ovarian tumours; in a uterine cyst, however, the cervix, or the region near the cervix, is often involved. There are, however, fallacies about this symptom, as an ovarian growth burrowing under the peritoneum may appear on touch to involve the cervix. Bilateral solid tumours of the ovary have often been taken for multiple pedunculated myoma of the uterus. In the ovarian affection, however, the uterus will be found on careful bimanual palpation to be completely free from both tumours.

*Parovarian cysts* are more flaccid than true ovarian cysts, and their



walls are clearly very thin. Tapping for diagnosis is not justifiable. There is no certain method for diagnosing a pathologically ovarian from a pathologically parovarian cyst when either burrows in the broad ligament. In both cases the uterus may be drawn up very high on the front of the tumour. *Dilated Fallopian tube* seldom rises high above the pelvic brim. Whether a hydrosalpinx or a pyosalpinx it is nearly always associated with a distinct history of pelvic pain and inflammation, the pain often subsiding whilst the tube distends. An ovarian cyst rarely gives pain at first, and often causes suffering when large. The dilated tube curls behind the cervix, its extremity lying in Douglas's pouch.

*Pregnancy* must be always borne in mind, more especially when complicated with *hydramnion*. Yet experienced authorities have taken even simple pregnancy for ovarian tumour. Remember that in cases of malignant ovarian disease in young girls amenorrhœa often sets in early, and the tumour grows quickly.

*Multiple hydatids of the omentum* feel, through the abdominal wall, like potatoes; they are clearly separate, unlike the loculi of an ovarian cyst. They are also unconnected with the pelvic viscera, but there are sometimes hydatids in the pelvis also. *Hydatid of the liver* and *dilated gall-bladder* must be remembered when a cyst is detected in the right side of the abdomen.

Real difficulty may be encountered when an ovarian tumour, especially a cyst, lies displaced and adherent in one flank, or in the epigastrium. In the first case *renal tumour* may be suspected. This tumour, however, develops posteriorly and superiorly, and grows downwards and forwards, and the colon lies in front of it; but adherent gut sometimes lies in front of a laterally displaced ovarian tumour. A small dermoid or simple ovarian cyst lying near the epigastrium or behind the umbilicus must be distinguished from a *cyst or solid tumour of the pancreas*, where marked special clinical symptoms are usually present.

*Phantom tumours* are resonant on percussion, especially if the patient be well purged before examination. The abdominal muscles contract firmly when touched, so that a solid tumour may be suspected. On bimanual palpation under anæsthesia the fingers of both hands can be made to meet, no tumour lying between them, and the uterus and appendages feel free from disease. *Distended bladder* must not be overlooked.

The nature of *ascites* and its morbid associations are well known. When the patient lies on her back there is resonance anteriorly and dulness in the flanks; when she lies on one side there will be resonance over the opposite or upper side, whilst in ovarian cysts the dulness lies anteriorly, and there is usually clear resonance in both flanks in any position. In constipated subjects with an ovarian cyst one or both flanks may be dull on percussion. Adherent gut in front of the cyst will be resonant. *Encysted dropsy* (anterior) is associated with anterior dulness and resonance in the flanks, as is *cyst of the urachus*. The former disease is generally associated with tubercular peritonitis or pelvic inflammation, the latter is very rare. The urachal cyst lies over the middle line between the umbilicus and pubes, and is quite fixed. When it discharges urine through the umbilicus it betrays its character at once, though in one instance (Unterberger) it was taken at first for rupture of an ovarian cyst through the umbilicus.

*Solid hepatic and splenic tumours* and *solid tumours of the mesentery* and *spleen* ought not to be mistaken for solid ovarian tumours. The pelvic organs will be found free on examination. But complications often deceive.

*Treatment of Ovarian Tumours.*—An ovarian tumour should be removed



when diagnosed. The author has referred, at the beginning of the paragraphs on "Symptoms of Ovarian Tumours," to cases detected very early; the surgeon must not be in a hurry to remove an ovary which may be simply swollen through inflammation. He has also devoted a former paragraph to "Ovariectomy and Malignant Disease." Tapping is very objectionable, and only justified in the case of a patient subject to a large cyst and attacked with acute pulmonary disease, the cyst causing dangerous dyspnoea. Thoracic visceral disorders and renal or hepatic disease are not contra-indications to ovariectomy unless they be advanced, for they are aggravated by the presence of the tumour. Even diabetic subjects bear ovariectomy, but only if carefully treated for a month or two first. Ovariectomy during pregnancy is highly successful, though it demands special care, whilst ovarian cysts are apt to undergo serious changes in the puerperium. In short, there is no treatment for ovarian tumour but ovariectomy.

OVARIOECTOMY.—The general principles guiding the surgeon in abdominal operations will be found under the proper headings in this Encyclopædia. Ovariectomy is in some cases an operation of extreme ease and safety. In others, especially when the tumour is malignant or dermoid and strongly adherent, it may be very difficult and fatal in spite of the most skilful operating and the most careful after-treatment.

It is best, but not absolutely necessary when there is urgency, not to operate during menstruation. The patient should be placed in the Trendelenburg posture whenever it is certain or probable that any part of the tumour is connected by adhesions, or by burrowing, with the pelvis. When it is clear that the tumour is chiefly one large cyst, with healthy fluid contents, a three-inch incision through the middle line is quite sufficient for its extraction after tapping. Under other circumstances the abdominal wound should be made larger, nor should the surgeon scruple to enlarge it further in the course of the operation if necessary. He must make sure that he has opened the peritoneum, and not taken it for a cyst wall. Strong anterior parietal adhesions demand great care during separation. The tapping of a cyst is not difficult, but loculi may require breaking down with the hand. This manœuvre must not be done too slowly, as it causes hæmorrhage into the cyst. Sponges should be packed in the peritoneal cavity around the trocar, especially if there be the least suspicion of infected cyst contents. The collapsed cyst or the solid tumour must be carefully freed from adhesions before complete extraction. Detached omentum should always be ligatured, else it may ooze, and oozing encourages sepsis. When in separating adherent intestine its serous coat is torn, that coat must be sutured. Small lacerations laying open the intestinal canal may be safely treated by sutures carefully applied; but extensive damage will require resection of the injured gut.

The enucleation of a sessile tumour requires the surgeon to bear in mind the anatomy of the pelvis. The fundus uteri is the best landmark, and the peritoneal relations of the large intestine to the tumour must be carefully inspected. There is often much oozing from the capsule which requires ligature of vessels, pressure by sponges, and, in bad cases, drainage by tube or gauze.

When there is a pedicle, the outer border, containing the ovarian artery with its big veins, should always be tied separately. The pedicle is then transfixed, a loop of No. 3 silk being passed through it. The loop is cut, the two silk threads thus formed are crossed on one side. Then the ends of one thread are tied well into the groove made by the ligature of the ovarian vessels, and the ends of the other thread tied close to the corner of



the uterus. Sometimes a double transfixion is necessary. In very wide pedicles the inner like the outer border should be tied separately. The pedicle must always be relaxed whenever a ligature is being tied.

The free use of the pressure-forceps to bleeding points on the parietal peritoneum, etc., is specially advisable in ovariectomy. It avoids the leaving of too many ligatures in the peritoneal cavity; but great care must be taken lest a forceps be left in that cavity.

Flushing of the peritoneum, closure of the abdominal wound, drainage, and after-treatment are conducted according to the general rules of abdominal surgery. Drainage is seldom necessary. Flushing, or "irrigation" with saline solution is never needed in a simple case; but it is very beneficial in bad cases where there is shock, and where much enucleation or separation of adhesions is practised. In such cases two or three pints of the solution should be left in the peritoneal cavity.

*Results of Ovariectomy.*—The operator, should he be in a position to perform ovariectomy on a naturally assorted series, may attain a mortality of under 5 or 6 per cent. Under certain conditions, however, the chance of death must always be strong. Amongst the most unfavourable cases are adherent dermoids, where the fatty and solid contents have partially invaded adjacent viscera and peritoneum, and malignant tumours closely united to intestine. In Olshausen's practice the mortality of ovariectomy for malignant tumour was 28 per cent.

COMPLICATIONS FOLLOWING OVARIOTOMY.—As this operation once took the lead in abdominal surgery, much that applies to that branch of our art was for long too exclusively associated with ovariectomy. Many too well-known complications are in no sense special effects of the removal of ovarian tumours. Hence for fuller details on sapræmia, septicæmia, peritonitis, and intestinal obstruction, the reader is referred to their proper headings.

*Hæmorrhage and Hæmatoma.*—Internal hæmorrhage within twenty-four hours after ovariectomy is the result of slipping of the ligature, excepting, perhaps, in a very few cases where a large vein is wounded during transfixing. It has been known to follow the operation alike where the loop-knot, the double interlocking-knot, the Staffordshire knot, and the ligature of both sides of the pedicle separately without interlocking, have been practised. It may be that in one or two instances a ligature has snapped after the completion of the operation; but as a rule it is the pedicle that slips from under the knot. The accident is due either to faulty tying of the knot, cutting of the pedicle too short, or most probably to dragging on the pedicle too much during ligature. When the pedicle is relaxed by the assistant the operator often fails to pull the knot tight enough, especially when the parts sink after relaxation deeply in the pelvis, so that the ends of the silk are badly placed for manipulation. Manœuvres with sponges, etc., deep in the pelvis, at the end of the operation, have sometimes caused the ligature to slip unobserved, or have torn one border of the pedicle.

The symptoms are those of internal hæmorrhage, usually very marked. They come on from about two to twelve hours after the operation, and there is often the familiar remission for a time after each attack of syncope. No treatment is of avail, but the immediate reopening of the wound, securing of the pedicle with forceps, clearing out clots and fluid blood, and religature of the pedicle. Infusion of saline solution may be necessary.

*Hæmatoma of the broad ligament* is the result of wounding of a big vessel by a too sharply pointed needle during transfixion and ligature. This complication may cause fatal hæmorrhage by displacing the ligature,



but a more frequent result is suppuration of the clot during the second or third week, probably from its proximity to the rectum. The symptoms are occasionally mistaken for parametritis. Sometimes a vaginal incision is necessary.

Pure *septicæmia* is occasionally seen, especially after the removal of suppurating adherent dermoids or malignant tumours extremely adherent to small intestine. It is marked by a considerable rise of temperature, a great rise with weakening of the pulse, and the appearance of the bad general symptoms of septic infection. The urine becomes albuminous, and is at length suppressed. The tongue not rarely remains moist to the last, abdominal distension and retention of the flatus may be trifling or absent, and there may be little or no sickness. The patient may tolerate fluids by the mouth, but the stomach neither rejects nor absorbs them. The above combination of symptoms is of the gravest importance.

Septicæmia is usually associated with *peritonitis*, recognised by three symptoms variable in relative degree—vomiting, tympanites, and retention of flatus. The tongue soon gets dry. Sometimes peritonitis comes on during the second or third week. Acute septic peritonitis is a trifle less fatal, and is much more manageable than pure septicæmia. The ejection of green or dark vomit should be encouraged by doses of warm water and soda, the patient should be fed by enemata, and above all the passage of flatus promoted. For this purpose gruel and turpentine enemata are efficacious. Saline purgatives are too often vomited. When flatus comes away freely the special symptoms of sepsis nearly always diminish in proportion.

INTESTINAL OBSTRUCTION is frequently seen in the form of pseudo-ileus, paralysis, and distension, with kinking of coils of gut. This condition is intimately associated with sepsis and peritonitis. Acute mechanical obstruction sometimes sets in during the second or third week, but is oftener a remote result of ovariectomy. Intestine is apt to adhere to the pedicle, or the omentum gets attached to the pedicle and indirectly causes obstruction. Early obstruction has been relieved by operation; but the operator must be careful about diagnosis, as secondary operations are very unsuccessful in cases where pseudo-ileus exists, while enemata and purgatives not rarely save the case.

*Instruments, etc., left in Peritoneal Cavity.*—This accident is very common in abdominal section, and especially ovariectomy, where enucleation, the separation of vascular adhesions, and the securing of bleeding points require the use of many sponges and forceps. Fortunately, the oversight is often recognised just as the patient has been put to bed, then reopening of the wound and removal of the foreign body involves but little danger. Trustworthy and experienced assistants and nurses may make mistakes through confusion, rather than carelessness. The unobserved loss of a forceps or sponge down a sink during operation causes intolerable anxiety. Schauta, on two occasions, reopened the peritoneum for a pad that had not been left there. In one case the operator's signet ring was left in Douglas's pouch, whence it was removed through the vagina. Neugebauer has collected no less than 108 cases of instruments left behind after abdominal section, from reports by unusually candid operators. His statistics show that the foreign body should be extracted directly it is missed, the mortality if it be left alone being very high. Out of 19 cases of pressure-forceps left behind, 7 died, 6 shortly after operation. Sponges do deadly harm; out of 29 cases the sponge was found at the post-mortem in 19. Out of 31 cases, where the foreign body was a gauze pad or compress, the gauze was found after death in 7. When the patient lives unrelieved the fate of the foreign



body is very uncertain. Sponges and drainage-tubes have slipped out of the abdominal wound at a dance, etc. A pressure-forceps has been passed at stool in 3 cases. As a rule the foreign body gets into the intestinal canal.

PARALYSIS is rare, and seems nearly always due to embolism. In Schramm's case the pelvis was elevated at the operation, the patient was single and aged 44, the tumour a small adeno-carcinoma. Facial palsy and paralysis of the left arm occurred on the second day and gradually passed off during convalescence (*Trans. Dresden Gynec. Soc.* Oct. 1899).

THROMBOSIS.—The external saphenous vein is apt to get plugged if the patient leaves her bed too soon; the leg then swells considerably. Rest, bandaging, and elevation of the limb usually effect a cure. When thrombosis sets in early after a deep enucleation, or after any other complicated ovariectomy, the progress is very grave. At the worst thrombosis is uncommon after ovariectomy, and much more frequent after any form of hysterectomy.

TETANUS.—Sixty-four cases of this complication were collected and tabulated by John Phillips in 1892. Martin and others have reported a few cases since that date. Asepsis is the true prophylactic against tetanus.

RASHES are due to irritating perspiration, the contact of blankets against the skin, and soap and water enemata. The mottled rash in septic cases is a grave symptom. *Jaundice*, purely catarrhal, occurs in some patients during convalescence. When associated with septic symptoms it is extremely serious, but simple sallowness of the skin in mild septic cases is often mistaken for jaundice.

PAROTITIS occasionally follows ovariectomy and other abdominal sections. Stephen Paget, Martin and Rüttermann and others, have written on this complication. In a case in the author's practice the tongue was foul, and there were carious molars on the affected side. No suppuration occurred. In some cases this complication ends in periostitis and necrosis of the inferior maxilla.

ACUTE MANIA has been observed after ovariectomy, but its relative frequency after that operation has been exaggerated. Barwell, Pozzi, Gaillard Thomas, and Butler-Smythe have written on the subject. As a rule the symptoms are mild, and unless the patient is predisposed to insanity ultimate cure is complete. Halliday Croom reports a case where the symptoms appeared on the third day, and death took place on the sixth. The operation was perfectly simple, the opposite ovary was healthy, and therefore not removed, and there was no evidence of sepsis. A distant relative was weak-minded. Mania follows other operations; it is not certain in these cases how far it is due to shock, and how far to removal of a particular organ, the ovary.

TUMOURS OF THE OVARIAN LIGAMENT.—This ligament includes fibrous and muscular tissue, and anatomically and pathologically belongs to the uterus, but it may contain detached pieces of ovarian substance; the significance of this fact has been noted in the paragraphs on the physiology of the ovary. Clinically and surgically tumours of this ligament are ovarian rather than uterine. True fibromas of the ovarian ligament have been successfully removed by Doléris, Briggs, and the author. The tumour weighed over 16 lbs. in the author's case, the ovaries and uterus were free from any new growth. This case (and possibly others) has been included amongst pedunculated tumours of the broad ligament in tables prepared by Gross, Vautrin, and Lang. Penrose (New York) removed a tumour which he reports as a sarcoma of the ovarian ligament, it was stony hard



(like a fibroma), and weighed 5 lbs. These tumours are easily removed, when large it is safer to take away the ovary as well, but in Doléris's case it was saved.

**BROAD LIGAMENT CYSTS.**—*Dilated lymph spaces* form irregular bullæ, yellowish in colour and thin-walled. They are lined internally with endothelium, and when the broad ligament is divided, drain away. They are clearly due to obstruction, and are often very prominent in cases of large uterine fibroid. The author has twice opened a cyst in the broad ligament, which he found to contain several pints of a lymph-like fluid. In each case the cyst burrowed deeply into the broad ligament below the ovary, and was lined with endothelium, or rather consisted of endothelium lining a cavity, which was the result of the dilatation of a lymph space. Drainage was necessary in both cases, as the base of a cyst of this kind cannot be extirpated.

The *parovarian* or *simple broad ligament cyst* is much more familiar both to the operator and the pathologist. Its histogenesis has been and is still much disputed. Most observers consider that, like papillomatous cysts of the ovary, it arises from relics of the mesonephron. The parovarian cyst, however, arises from those relics which lie between the folds of the broad ligament, and constitute the parovarium. But Kossmann's theory has been noted already in the paragraph on "Histogenesis of the Papillomatous Cyst." He insists that the simple parovarian cyst, free from any trace of papillary growth, has nothing to do with the tubes of the parovarium, but rises from Müllerian elements like the Fallopian tube. These relics ("Nebentuben," accessory tubes) sometimes develop as cystic cavities, lined like the tubal canal with ciliated epithelium, and enlarging become what is, falsely in his opinion, known as parovarian cysts. Sometimes papillomata grow from the epithelial investment, and thus, in Kossmann's opinion, originates the papillomatous cyst of the broad ligament independent of the ovary. The present author wrote in 1884 that "it might be contended that some of the minute non-parovarian cysts are developed from Müller's duct, which ultimately becomes the Fallopian tube. There is no evidence, however, that any true Fallopian cyst has ever been formed." Kossmann, on the other hand, declares that there is not the slightest evidence that a "parovarian" cyst has ever been actually traced from a dilated parovarian tube. The development of a cyst of this kind from its earliest stage is difficult to note with accuracy. There is need of further research.

When large the parovarian or simple broad ligament cyst is a very characteristic growth. It occupied the greater part of the mesosalpinx, so that the Fallopian tube and its fimbriæ are stretched over its upper surface, whilst the ovary generally hangs freely from its lower surface, but sometimes is stretched or more often extremely flattened through pressure. Occasionally (not so often as in the case of papillomatous cyst of the broad ligament) this cyst burrows into the mesometrium.

The wall of the parovarian cyst is often very thin, but proportionately tough and fibrous. It is lined with epithelium, often cubical from pressure, but originally, there can be little doubt, columnar and ciliated. It is almost invariably unilocular. It contains a clear watery fluid, often to the amount of several pints, of very low specific gravity, rich in chlorides, but free from albumin. It can be peeled entirely out of the folds of the broad ligament. It is liable to rupture, in which case there is little or no local or constitutional disturbance, indeed spontaneous cure may ensue. The same has followed tapping.

*Symptoms and Diagnosis.*—The parovarian cyst often causes nothing but physical symptoms, which last for months without complications. A flaccid or moderately tense cystic swelling develops and rises above the



pubes. It is freely movable, and the uterus is quite distinct; in many cases the ovary can also be felt distinctly below it; this proves its true nature. It is very common; many cases where the ovary is flattened out on its surface have been registered as "ovarian." If neglected it may set up peritonitis or may suppurate, and it is certainly apt to undergo papillomatous degeneration.

*Treatment.*—Keith used to *tap* parovarian cysts, and in many cases permanent cure ensued. But a cyst of this kind has been tapped seven times, and then had to be removed; it is, too, a morbid growth best taken away, as not rarely it contains papillomatous elements. Its removal is easy and successful, whilst the extirpation of a papilloma is difficult, dangerous, and not rarely followed by recurrence. It should be removed, therefore, by ovariectomy—that is to say, it must be tapped after exposure through the abdominal wound, drawn out of the abdomen, and its pedicle ligatured and then divided. This pedicle is the same as in ovariectomy. The author has in several cases, where the patient was young, enucleated a parovarian cyst through an incision in the mesosalpinx, afterwards closed by suture. The tube and ovary were thus spared. When the cyst burrows into the mesometrium, below the level of the ovary, enucleation cannot be avoided. There is often much oozing, which must be checked.

*Papillomatous cyst of the broad ligament* is very similar to papillomatous ovarian cyst already described, but the ovary is uninvolved. Its histogenesis is practically the same, and it certainly may develop from a simple parovarian cyst. It burrows very deeply under the broad ligament, and pushes the uterus far upwards and forwards. It is never freely movable, often fixed, and always lies chiefly on one side. It is apt to produce pain from pressure. By the above characters it may be diagnosed; in shape it is usually oval. It is often bilateral. If left alone it ultimately ruptures, and its papillomatous contents soon become disseminated, with fatal results.

This kind of cyst must be removed directly it is diagnosed. It must be carefully and thoroughly enucleated from the broad ligament, hæmorrhage from which must be stopped by the free use of pressure forceps. Sometimes this process proves easy, as a rule it is difficult, much blood is lost, and papillomatous masses are often left behind deep in the pelvis. The ureter may be torn or cut. In bilateral cases total removal of the uterus with the cysts may be required; the danger of damaging the ureters is, however, very great if that radical operation be done. The patient should always be placed in the Trendelenburg posture when a papillomatous cyst of the broad ligament is removed, so that plenty of light may be turned on to the pelvic cavity.

*Solid tumours of the broad ligament* are noted under "Broad Ligament, Diseases of the." "Fibroid" or myoma of the ligament is really distinct from uterine "fibroid," but is usually classed with that well-known morbid growth.

*Primary dermoid cyst of pelvic connective tissue* is closely allied to the retroperitoneal dermoid which has been found in males (Ord and Sewell, Lexer). It is like to ovarian dermoid cyst, and non-malignant, unlike ovarian teratoma. It of necessity tends to invade the mesometrium, not the mesosalpinx, and burrows as far as the perineum or upwards into the abdomen. Beyea has collected notes on 21 cases. In his own case the patient was 38; both her ovaries had been removed twelve years previously, the catamenia ceasing at once. He enucleated the dermoid from the right broad ligament, but hæmorrhage could not be controlled until he amputated the uterus above the cervix. The patient recovered. Page of Newcastle operated on a woman aged 47; the cyst reached to the umbilicus, and lay behind the rectum. He made a semilunar incision 6 inches long, its centre



lying midway between the anus and coccyx. After extracting hair and putty-like material Page successfully enucleated the cyst. Since that operation (1891) several tumours of that class have been removed through a perineal incision, and several, including Beyea's case, by abdominal section. Page relates that he was able to pass his hand and the greater part of his forearm through the perineal wound into the cavity of the cyst.

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### Primary Ovarian Pregnancy

The possibility of this condition is now admitted by several authorities, who till recently insisted that all ectopic pregnancies were primarily tubal. Van Tussenbroek, after careful examination of an ovary removed by Kouwer, found that a foetal sac of the size of a cob-nut lay on its surface. This sac held a minute foetus with amnion and chorion complete. British and foreign authorities who have examined this specimen agree with Van Tussenbroek's interpretation of its nature. The rupture of the sac caused severe intraperitoneal hæmorrhage, though the pregnancy was under the sixth week. Patenko's case, described many years earlier, was probably genuine. Anning and Littlewood's case, reported in January 1901, is of high clinical interest. The patient underwent operation for rupture of the sac. About two pints of fluid blood and clot were removed, and an ovum no bigger than a large pea was detected amidst the clot. There was a rent in the right ovary; into this the ovum and its sac exactly fitted; the corresponding tube was normal. The patient recovered. The ovary is preserved in the museum of the Royal College of Surgeons of England. Another authentic case was reported in 1900 by Octavius Croft of Leeds. The sac and its contents, found entire, were removed by abdominal section; it ruptured during extraction and much hæmorrhage ensued, difficult to check; the patient died within a few days. The sac before rupture contained an ovum of about the fourth month with placenta, membranes, and amniotic fluid; the relations of the sac were those of an ovarian cyst with a short pedicle; the Fallopian tube was healthy. Hastings Gilford's alleged case is still under examination.

Thus it seems evident that primary ovarian pregnancy may occur, and that as a rule it is most probably arrested at a very early stage (Van Tussenbroek, Anning and Littlewood), yet may continue at least to the fourth month (Croft). The bleeding from the rupture may be very severe, even very early in pregnancy (Kouwer, Anning and Littlewood), whilst when gestation is more advanced and the sac is capacious, its connections are full of large vessels, so that severe hæmorrhage will occur when it is extirpated (Croft). Some cases reported many years ago as "ruptured ovarian apoplexy" may have been primary ovarian pregnancies.

When the pregnancy is advanced it is not easy to prove that it was primarily ovarian. If the corresponding tube be normal, it does not follow that the ovum did not originally develop in its canal, for when the tube expels an ovum out of its ostium it rapidly regains its natural appearance. The ovary is occasionally flattened out widely over the wall of a big extra-uterine sac, but that is no proof that the sac arose from the interior of that organ. (See also "Extra-Uterine Gestation," vol. iii.)

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**Overlaying.** See MEDICINE, FORENSIC, vol. vii.

**Oxalic Acid Poisoning.** See TOXICOLOGY.

**Oxaluria.** See URINE.

**Oxygen** is a colourless, odourless, and tasteless gas, somewhat heavier than atmospheric air, which contains 20·96 volumes per cent of this gas. Physiological experiments prove that, on inhalation of an atmosphere of pure oxygen, the normal animal organism does not take up more oxygen than on inhalation of the ordinary atmospheric air, or in other words, that the atmospheric air is sufficient for the oxidation of nearly all the reduced hæmoglobin passing through the lungs under normal conditions.

At the present time there is considerable divergence of opinion as to the therapeutic value of oxygen, but in spite of theoretical objections its administration is beneficial to some patients in whom dyspnœa and cyanosis are urgent symptoms, and especially when these symptoms are dependent on some obstruction of the respiratory passages. Thus in some cases of true asthma, bronchitis, and laryngeal diphtheria, and in cases in which there is pressure on the trachea or extrapulmonary bronchi, inhalation of an atmosphere rich in oxygen is useful in reducing the frequency of the pulse and respirations and in relieving dyspnœa and cyanosis. In pneumonia, pulmonary emphysema, and cardiac disease, the benefit of oxygen inhalation is less evident. The improvement obtained in some of the latter cases is frequently ascribed to mental suggestion, but is often largely if not entirely due to the stimulant action of the gas. In most of these cases the use of oxygen induces only temporary improvement, rarely, if ever, is the improvement permanent. As a stimulant, oxygen is also useful in severe collapse, in excessive chloroform narcosis, and in carbon monoxide, opium, and other forms of narcotic poisoning.

Oxygen inhalations have probably no influence either on the fixed tissues or the metabolism of the organism, and as a rule no improvement has been obtained by the use of oxygen in diseases such as anæmia, pernicious anæmia, leucocythæmia, tuberculosis, diabetes, gout, and other constitutional diseases. The gas should be administered in a concentrated form, and hence through a mask, if a stimulant effect is desired. If the inhalation, whether continuous or interrupted, is to be prolonged over a considerable period of time, it is advisable to connect the tube leading from the oxygen cylinder with a wash bottle so that the gas may pass through water before being inhaled.

Oxygen is also employed on account of its local oxidising and antiseptic action. Water saturated with oxygen has long been employed by some both for drinking and balneological purposes, but the pure gas has no action on the skin itself. The local application of oxygen is more readily obtained by the use of hydrogen peroxide, which on contact with the tissues is decomposed into water and oxygen. The local application of the watery solution of hydrogen peroxide is therefore useful in such conditions as suppurating wounds, septic ulcers, diphtheria, and tonsillitis.



**Ozæna.** See NOSE.

**Pachymeningitis.** See MENINGES AND SPINAL CORD.

## Palate.

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ANATOMY.—A short reference to the anatomy of the palate is necessary before entering upon the consideration of its abnormalities and diseases.

The *hard palate* has normally the shape of a regular arch, but varies considerably in different individuals. It is composed of an osseous framework covered by periosteum and mucous membrane. The osseous portion, which consists of the two horizontal planes of the superior maxilla and of the palate bones, is roughened on its under surface for the attachment of the muco-periosteum, perforated by foramina for the transmission of its nutrient vessels, and has attached to it posteriorly the palatine aponeurosis. It receives its blood-supply from above as well as below. The relations and component parts of the intermaxillary bone are considered under Hare-Lip.

The mucous membrane of the hard palate is dense, contains numerous small glands, and is closely adherent to the periosteum. Its blood-supply is derived from two sources—the *posterior palatine artery*, which, after emerging at the posterior palatine canal, situated internal to the last molar tooth, runs forward in a groove in the bone parallel to and about  $\frac{1}{2}$  to  $\frac{3}{4}$  of an inch internal to the alveolar border, and the *naso-palatine artery*, which descends through the anterior palatine canal. The terminal twigs of these vessels anastomose with each other.

*Nerves.*—The *soft palate* consists of an aponeurosis firmly attached to the posterior border of the hard palate, of fibrous tissue, of muscles, and of mucous membrane. From its side there pass off two folds of mucous membrane, the *pillars of the fauces*, the anterior of which descends to the tongue and the posterior to the pharynx. Posteriorly, the soft palate terminates in the uvula.

The following are the muscles of the soft palate :—

The *palato-pharyngeus* begins in the median raphe in two layers which enclose the levator palati and azygos uvulae ; it then passes downwards and backwards in the posterior pillar of the fauces and spreads out into the side of the pharynx and along the posterior border of the thyroid cartilages. *Action.*—It elevates the larynx and approximates the posterior pillar of the fauces.

The *palato-glossus* blends with its fellow of the opposite side on the anterior surface of the soft palate, passes down in the anterior pillar of the fauces, and is inserted into the side of the tongue. *Action.*—It draws the soft palate downwards and elevates the side and the base of the tongue.

The *levator palati* arises from the under surface of the petrous bone and from the lower border of the Eustachian tube, passes downwards, and is inserted into the median raphe between the two layers of the palato-glossus. *Action.*—It elevates the soft palate and brings it in contact with the posterior wall of the pharynx.



The *tensor palati* arises from the scaphoid fossa of the internal pterygoid plate and from the Eustachian tube. It ends in a tendon which is reflected round the hamular process and is inserted into the ridge on the under surface of the palate bone and into the anterior surface of the soft palate. *Action*.—It renders the soft palate tense and opens the Eustachian tube during deglutition.

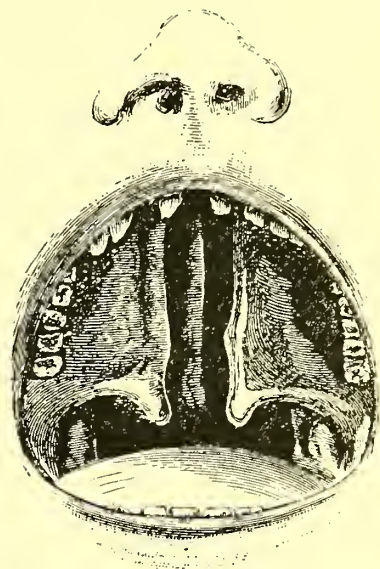
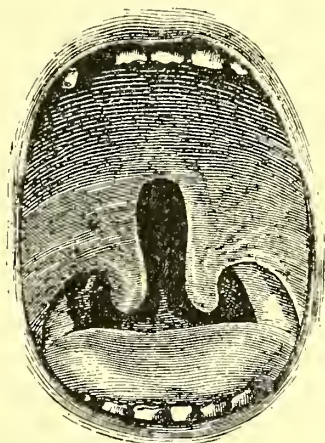
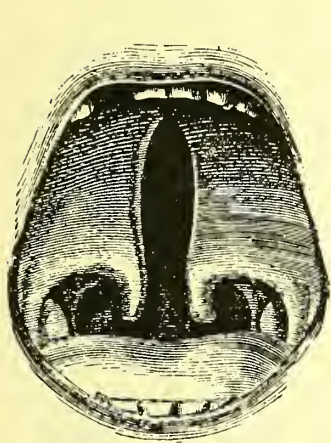
The *azygos uvulae* arises from the posterior nasal spine and descends into the uvula. *Action*.—It elevates and shortens the uvula.

The *mucous membrane* on the posterior surface of the soft palate is thin and continuous with that of the nose; on the anterior surface it is thick and contains mucous glands.

The *blood-supply* is derived from the posterior palatine, the ascending palatine, and the ascending pharyngeal arteries.

*The Nerve-Supply*.—*Sensory*. Glossopharyngeal fibres of superior laryngeal branch of vagus.

*Motor*.—N. Accessorius. Glosso-pharyngeal.



FIGS. 1 and 2.—Various degrees of simple fissure of the palate. (Mason.)

FIG. 3.—Complete cleft palate without alveolar or labial deformity. The vomer is separated from the lateral segments. (Mason.)

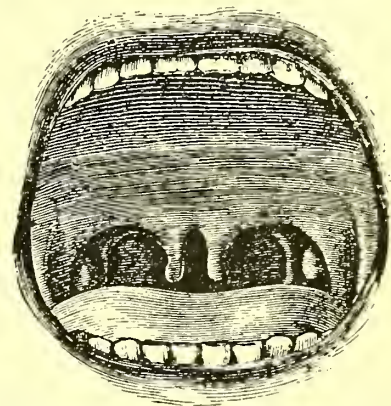
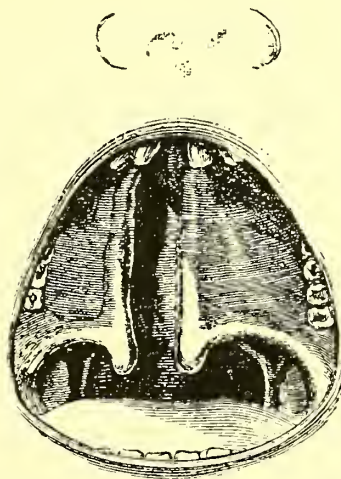
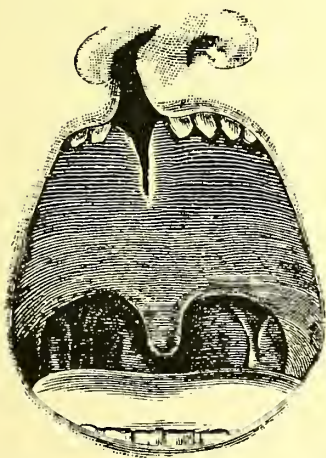


FIG. 4.—Unusual form of cleft involving the alveolar arch and the anterior portion of palate only. (Mason.)

FIG. 5.—Complete unilateral cleft palate without alveolar deficiency. The vomer is attached to the left palatal segment. (Mason.)

FIG. 6.—Simple fissure of the palate. (Mason.)

**CLEFT PALATE.**—Acquired defects are described under diseases of the palate.

The manner in which the palate is developed determines the variety of the cleft. The palatal processes grow inwards and unite in the middle line from before backwards, the vomer joins the hard palate from above, the intermaxillary bone terminates at the anterior palatine canal.

*Extent*.—The cleft may involve merely the soft palate and vary in degree from little more than a bifided uvula to a complete fissure of the velum.



The entire soft palate may be involved, and the hard fissured in part or up to the anterior palatine canal.

In the severe forms the cleft extends beyond the palatal processes, and is complicated by the presence of a unilateral or double hare-lip.

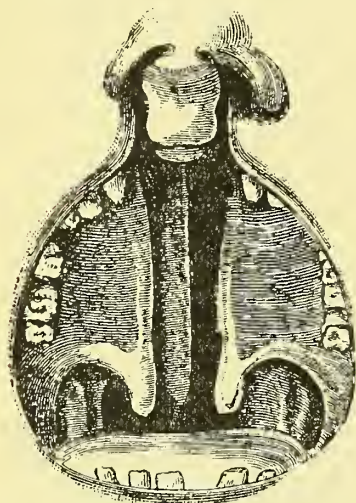
*Appearances.*—Usually the cleft is mesial, or it may appear to be unilateral in consequence of the union of one segment, generally the right, with the medium septum; in these cases there is often a left-sided hare-lip.

The vomer, if well developed, may project between the palatal processes, and the cleft appears to be double. When the fissure extends beyond the palatal processes it diverges at the anterior palatine canal to pass forward through the sutures of the intermaxillary bone, usually between the endognathion and mesognathion.

*The width* of the cleft varies greatly; in some cases the segments of the hard palate are more or less horizontal, in others they may run almost vertically upwards.

In addition to those described above, other congenital defects are occasionally met with in the palate. The deformity may be limited entirely to the anterior part of the hard palate with or without an associated alveolar cleft; it may exist as an oval defect at the junction of the hard and soft palate, in the hard or in the soft palate only, or the continuity of a cleft may be interrupted by a bridge of tissue. Sometimes one or two vertical clefts occur in the anterior pillar of the fauces near where it joins the tongue.

FIG. 7.—Complete cleft palate and double alveolar hare-lip, simulating a double lateral cleft due to the vomer being seen free between the palatal segments. (Mason.)



*Effects.*—The results of cleft palate have to be considered in their relations to nutrition, to the mucous membrane, and to speech.

As regards nutrition, an infant with a well-marked palatal deficiency is unable to suck, swallowing is interfered with, fluids are apt to regurgitate through the nose; therefore, unless the child be carefully fed, its nutrition becomes seriously impaired, and it may die of starvation, or its physical development be impeded.

The effects upon the mucous membrane arise from dryness, produced by the abnormal exposure to air, and from infection. Children with cleft palate are frequently the subjects of chronic naso-pharyngeal catarrh, adenoids, and enlarged tonsils.

Articulation is indistinct and laborious. The nasal twang is characteristic. Speech is affected even when the aperture in the palate is small.

In adults the difficulty in swallowing has been overcome to a considerable extent, and the patient by carefully training himself may have effected a degree of improvement in his speech.

**TREATMENT OF CLEFT PALATE.**—Under this heading there have to be considered:—

(1) *Method of feeding* infants who are the subjects of cleft palate is described under Hare-Lip.

(2) *Treatment of the Deformity.*—Closure of the cleft by operation is the most satisfactory method. Mechanical appliances ought to be reserved for special cases which will be referred to later.

Before describing the operation for cleft palate it is well to consider the benefits to be derived from successful surgical treatment. At all ages, and even in adults, the patients are in a better condition after successful



surgical interference than when no operation has been performed. Food has not the same tendency to regurgitate through the nose, taste and smell are improved, and speech is more distinct.

As regards results, much depends upon careful attention to the essential details of the operation, upon the width of the cleft and the arch of the palate, upon the size of its segments, the amount of tissue available for the plastic operation, and upon the age of the patient. It must, however, be borne in mind that when union takes place from end to end the results, as regards speech, are sometimes disappointing. This is the case especially when the soft palate is scanty and rigid and does not effectively shut off the naso-pharynx, and also in certain cases of imperfect development of the nasal cavities.

The treatment of cleft palate does not cease with successful surgical treatment. The operation renders the patient capable of being educated to speak properly.

**PERIOD FOR OPERATING.**—When hare-lip and cleft palate are associated, the former ought to be closed as early as possible—*vide* Hare-Lip.

The earlier the cleft can be closed the better the results as regards speech. Partial clefts can be closed at an earlier period than complete fissures, which require a more extensive and prolonged operation, and entail greater loss of blood and more shock.

Some surgeons operate during the first few months of life, and others even as early as the fourth to fifth week.

If the soft palate alone be fissured the best period on the whole is between eighteen months and two years, although sometimes in suitable cases the operation may be done before the period of first dentition; when the cleft is complete, from the third year onwards. The tendency is, however, to operate in all cases at an earlier period than was formerly the case.

Age alone does not determine the date of operative interference. None the less important is the state of the child's general health, the condition of its mouth and naso-pharynx, and whether it is tractable or otherwise.

**ESSENTIALS FOR SUCCESS.**—Primary union is of the first importance. This is to be attained by paring the edges of the cleft in one piece, by completely separating up muco-periosteum flaps, by the division of all tense structures, by accurate suturing, by the prevention of septic infection, and by rest of the wound as far as possible. It is also most important that the sutured soft palate should be mobile.

*Preliminary Treatment.*—The operation not being one of emergency, the surgeon is able to choose his own time for operating. The best periods of the year are spring and summer.

The child ought to be under observation for some time. The possibility of its developing an infectious disease ought to be eliminated. A history of hæmophilia should be inquired for. The general health must be satisfactory, and no operation undertaken if the patient has recently recovered from any infectious disease or severe illness.

The child ought to be taught to breathe through its nose. After treatment is facilitated by accustoming the patient to take food from the side of a spoon or from a feeding-cup. The patient ought to be tractable and easily controlled by the nurse. It is advisable in the case of spoilt children not to have the mother with the child for any time until some days after the operation.

As in all operations in the mouth, asepsis should as far as possible be aimed at. Decayed teeth must be attended to, chronic pharyngitis



frequently requires treatment. The crusts are removed by bicarbonate of soda, 10 gr. to ʒj., and then boro-glyceride or some mild antiseptic brushed on. Adenoids and enlarged tonsils not infrequently require removal.

The patient should be on a light diet for a day or two before the operation, and the usual preliminaries as regards the stomach and bowels attended to prior to the administration of the anæsthetic. An enema of beef-tea and brandy may be given just before the operation.

*Instruments.*—Special gags, such as Smith's, Whitehead's, and other forms, are frequently used. The writer prefers any simple gag which will effectually keep the mouth open; an assistant depresses the tongue with a spatula. The following instruments are necessary: bistouries, sharp and narrow-bladed, also probe-pointed; scissors, straight and curved; long toothed forceps; periosteum elevators, straight and curved; dissecting forceps; sponge-holders, or what in many respects are better—Kocher's artery forceps, which securely grasp the sponges; cleft palate needles of various curves—those recommended by Rose are very satisfactory; silver wire, soft, pliable, and of moderate thickness, in lengths of 6 to 8 inches; silk-worm gut, horse hair, and sterile silk.

For stopping troublesome bleeding, pads of sterile gauze, ice, Horsley's wax, adrenalise chloride solution.

*The Anæsthetic.*—Chloroform is the most suitable, and if possible it ought to be administered by a skilled anæsthetist. Efficient administration of the anæsthetic is an important part of the operation. The patient should be well under before the operation is begun, and not allowed to come out until it is completed, lest vomiting should occur.

The chief assistant attends to the tongue and keeps the pharynx free from blood. Sponging ought to be done rapidly but lightly, and care taken not to bruise the tissues.

A useful adjuvant is the painting of the palate and pharynx with a 2 per cent solution of cocaine.

*Position of the Patient.*—The chest should be raised, and the head should be thrown back and hang over the end of the table. The surgeon sits or stands at the patient's head. Good light is essential.

*Extent of the Operation.*—The whole cleft ought to be closed at one operation, if possible. In some cases this cannot be done. The fissure in the hard palate is then dealt with first, and that in the soft subsequently closed, or, if that be impossible an artificial velum may have to be fitted.

**THE OPERATION.**—The operation described below is generally known as Langenbeck's. Avery of Charing Cross Hospital was the first in this country to completely close the cleft in the hard palate. Mr. Annandale, to whom the writer is indebted for many valuable hints in the treatment of hare-lip and cleft palate during the period he has been associated with him in the Edinburgh Royal Infirmary, performed, in 1864, the first successful operation in Scotland on the hard palate.

The operation for closure of the hard and soft palate consists usually of the following steps:—

- (1) Paring the edges of the cleft.
- (2) Detachment of muco-periosteum flaps.
- (3) Introduction of sutures.
- (4) Relief of tension.

The plan, practised by Rose, of detaching the flap before the edges are pared has much to recommend it.

(1) *Paring the Edges of the Cleft.*—If the vomer be attached to one side of the cleft the junction of the nasal and buccal mucous membrane should be divided.

The tip of the cleft uvula is grasped by a pair of long toothed forceps and pulled downwards and inwards so as to render the palate tense. The edge of the cleft is then pared from the apex to the portion of the uvula grasped by the forceps. The paring may be done from before backwards, or from behind forwards. Each side should be pared in one piece, and the apex of the cleft detached last; effectual paring is thus ensured. Too much tissue must not be removed, and the edges should not be bevelled.

(2) *Detachment of the Muco-periosteal Flaps.*—The bleeding after paring of the flaps is usually slight. An incision is then made with a sharp and rather stout knife, held perpendicular to the mucous surface. It begins on a level with, and just internal to, the last molar tooth, and is carried forward parallel, and not more than half an inch internal to, the alveolar border until it reaches the level of the lateral incisor tooth, if the cleft be complete, or, a little beyond the apex of the cleft if it be partial. The knife should divide the periosteum and pass down to the bone.

The incision, which is planned so as to pass external to the posterior palatine artery and to avoid the anterior palatine anteriorly, should be made close to the teeth. The former vessel can sometimes be felt pulsating, and avoided.

The muco-periosteal flap is now detached by raspatories of various curves, attention being paid to the following details. The points of the raspatories must be kept close to the bone in order to avoid working in the substance of or perforating the flap, which should be steadied by keeping the forefinger of the left hand on its buccal surface. The separation should extend inwards until the point of the raspatory can be protruded along the entire length of the cleft, forwards beyond the apex of the fissure, if it be partial, and just short of the anterior palatine canal if the fissure is complete, and backwards along the posterior border of the hard palate.

Bleeding is usually pretty free when the incision is made, especially if the posterior palatine artery be injured, but can be arrested by sponge pressure, unless the artery be partially divided, in which case the division should be made complete.

The operator proceeds with the separation of the flaps, while the assistant seizes every opportunity to remove the blood. Attention is now paid to the soft structures around the hamular process. This process is divided by some operators. The palatal aponeurosis attached to the back of the hard palate should be divided. The mucous membrane, reflected from the base of the soft palate into the nose, should also be divided. The division of these structures is effected by a probe-pointed bistoury and by curved scissors.

A pause can now be made to arrest bleeding and remove blood. In cases of complete cleft it is convenient to transfer the gag to the side of the mouth already dealt with, and to separate the other muco-periosteal flap.

When both flaps have been detached a raspatory should be introduced into the incision on each side and the flaps drawn together. If they fall readily into position, the next stage of the operation proceeded with; if not, any resisting spot is to be dealt with.

(3) *The Passage of Sutures.*—The method of passing the sutures varies with the part of the palate, the size of the mouth, etc.

It is sometimes possible, even in the hard palate, to pass a suitably curved needle threaded with silk first through one flap and then through the other. This direct method is employed in the soft palate and uvula.

The loop method is usually required when the hard palate is being dealt with. A needle of suitable curve threaded with silk a foot or more in length is passed through the whole thickness of the flap not less than one-eighth of an inch from the pared margin. The silk is caught at the eye of the needle by dissecting forceps or the point of another needle, and the needle withdrawn. A loop of silk is then left on one side of the cleft. The same proceeding is then repeated by

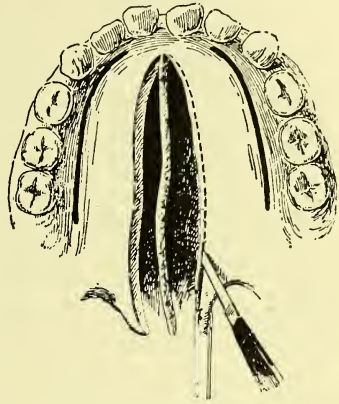


FIG. 8.—Paring the edges of the cleft. (After Cheyne and Burghard.)



passing the needle through the other flap at an exactly corresponding point. There are now two loops of silk, one of which may be passed through the other, which is gently withdrawn, and thus a loop passes through both flaps. Into this loop the bent end of the silver wire is caught, and pressed flat with forceps; the free ends of the silk are drawn upon and the wire carried through both flaps.

An equally convenient method is to pass one loop of silk, catch one end of the wire and draw it through one flap, and repeat the procedure on the other side with the other end of the wire. If this method be adopted care must be taken to prevent the wire kinking as its two ends are being drawn upon.

The first wire suture, which can be used for holding the palate forward, ought to be passed about the junction of the hard and soft palate, and farther from the edge of the cleft than the others, in order to act as a suture of relaxation at the part where failure is most likely to occur. The second is placed about the middle of the hard palate.

A third or fourth may be required.

The anterior part of the hard palate and the soft palate are best united by silkworm gut, and the uvula by horse-hair. Silk is not to be recommended.

The number of sutures required depends upon the extent of the cleft. They

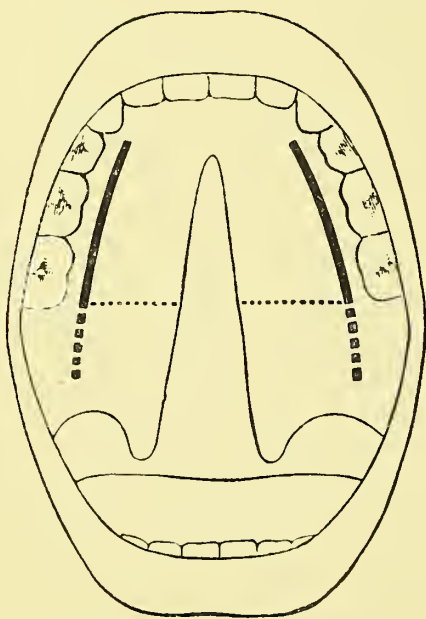


FIG. 9.—Extent of incisions.

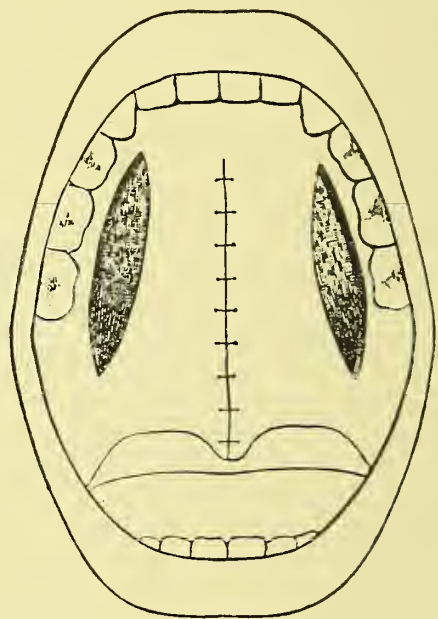


FIG. 10.—Operation complete.

ought not to be more than  $\frac{1}{4}$  inch apart in the soft and hard palates. Between the wire intermediate sutures of silkworm gut may be introduced. In a complete cleft the total number will vary from about eight to ten.

All the sutures ought to be introduced, and their ends clamped with forceps, before any of them are tied. The wire, if pliable, is tied in a single knot, and the ends cut fairly short and left flat across the line of union. Silkworm gut and horse-hair are tied by surgical knots. The sutures are conveniently tied from before backwards; before doing so, however, they are drawn together to see if the pared edges lie in accurate apposition. Care must be taken to avoid inversion of one edge and the uvula must be accurately united down to its very tip.

When the cleft extends up to the alveolar margin, in order to avoid damaging the artery a small gap may have to be closed at a subsequent period.

(4) *The Relief of Tension.*—This, the last, is not the least important stage of the operation. It is essential, not only for primary union, but also for the mobility of the soft palate, which ought to be sufficiently loose to move freely when the patient breathes. It may be said that the velum cannot be too loose. Sloughing is practically unknown.

An excellent rule is to divide with a probe-pointed bistoury or scissors any structure which interferes with the free movement of the velum.

In most cases it is necessary to prolong the lateral incision backwards by means of a probe-pointed bistoury through the soft palate just internal to the hamular process. Half the thickness of the palate ought to be divided, and the depth of the incision can if necessary be increased by the forefinger. If bleeding occurs pressure will usually arrest it.

Sometimes it is necessary to snick through the anterior and posterior pillars of



the fauces. As already mentioned, division of the mucous membrane passing from the soft palate into the nose must never be omitted.

When the soft palate alone is cleft the operation for its closure is known as *Staphylorrhaphy*. It differs from that described only in degree. The lateral incisions are not so extensive, and may be made after the sutures have been passed. The principles as regards the relief of tension are identical.

*After-treatment*.—The patient should be put to bed with the head low and turned to one side, so that blood and mucus will flow readily out of the mouth. The nurse or mother ought to be told that the child may vomit blood. If shock follow the operation, it is to be treated by appropriate measures.

*Feeding*.—No food is to be given for four or five hours at least, it being important to avoid sickness. For the first four or five days the patient is fed on milk and soda-water, beef-tea, or beef-jelly by means of a spoon or rubber tube attached to a feeder. Small quantities are given at intervals, say, of two hours. After the fifth day, custards, puddings, and soaked bread may be allowed. No solid food ought to be permitted for at least a fortnight.

The mouth is to be kept as clean as possible. After each feeding the child is to be given water to drink. The tendency of milk to remain in the mouth renders beef-tea and beef-jelly preferable. The wound may with advantage be swabbed with boro-glyceride two or three times a day if the child will allow it to be done.

The patient is to be kept as quiet as possible, and not allowed to put the fingers into the mouth. The palate should not be examined for a week. If the case follows a satisfactory course the patient may be allowed to get out of bed on the sixth day and be taken out of doors shortly afterwards.

*Removal of Sutures*.—The wounds at the side speedily granulate. The sutures are removed on the tenth to fourteenth day. They may be left in for a longer period. An anæsthetic is usually necessary in children. It is better to remove all the sutures at one time.

*COMPLICATIONS*.—*Primary hæmorrhage* occurs most freely when the lateral incisions are made, and sometimes after the incisions for relief of tension. The method of dealing with it has been described.

*Reactionary hæmorrhage* may occur in patients who have suffered from shock during the operation. Pressure and cold will arrest it. The posterior palatine canal may have to be plugged.

*Secondary hæmorrhage* is due to sepsis, and is rare. It may be serious. It is to be treated by cold pressure, clearing away the clots, and plugging the posterior palatine canal with a probe or Horsley's wax.

*Sepsis* is a serious complication as regards primary union. Loss of blood, rough handling, bruising with sponges, and a feeble constitution predispose. It is to be avoided by careful attention to the mouth before and after operation, and treated by a spray of peroxide of hydrogen, by boro-glyceride, and other mild antiseptics. It may be arrested, but generally leads to partial or complete failure.

*Failure* may be partial or complete. It is most likely to occur in wide clefts and when the palate is horizontal. The usual places for partial failure are at the junction of the hard and soft palate and anteriorly.

*Causes*.—In spite of every care failure will occasionally occur, but the risk is diminished in proportion to the attention paid to the preparation for, and essentials of, the operation.

*General Conditions*.—Measles, scarlet fever, thoracic or gastro-intestinal complications, severe colds.

*Local*.—Defective operating is the common cause—the essentials for success have already been sufficiently emphasised.—Crying, vomiting, the patient putting the finger in the mouth, sepsis.

*Treatment*.—Small holes, especially in the soft palate, frequently close of themselves. If union is doubtful the removal of the sutures ought to be delayed until the third week. A small opening may be made to close by a touch with a hot wire.

When failure is complete the sepsis ought to be dealt with. A second operation ought to be performed when the edges are granulating, usually in about a fortnight if the child be in good health. If the general condition



be unsatisfactory a further delay is necessary. Should the second operation fail a third attempt may be made four or five months later.

*Voice Training.*—It has been already pointed out that the treatment of cleft palate does not cease with a successful operation. The importance of voice training must be explained to the parents. Much care and patience is required, and the training, which has a twofold object—to develop the muscles of the palate by exercises and to correct the faulty habit of speech—has to be carried out for months, even years.

The former is best attained by making the patient take deep inspiration with the mouth open, while the tongue is kept in the floor of the mouth. Nose breathing ought also to be practised.

As regards speech, the subjects of cleft palate are apt to speak too quickly and to run their words together. This habit must be corrected.

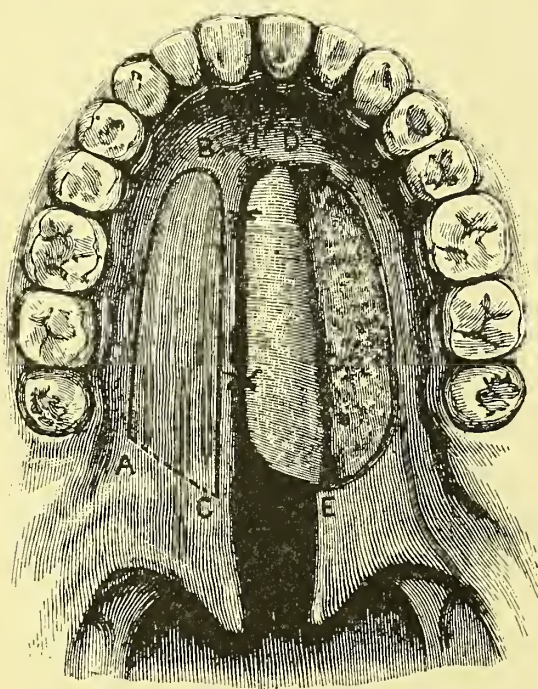


FIG. 11.—Davies Colley's operation, flap D, E, dissected up, turned over, and united to the opposite side with raw surface downward.

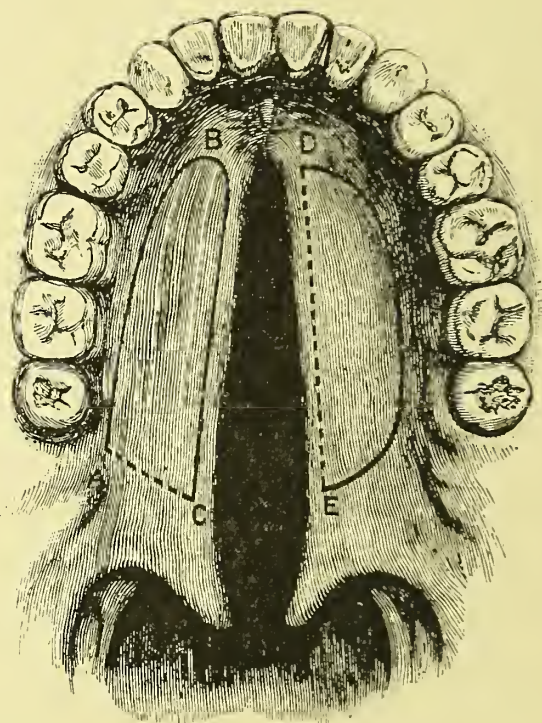


FIG. 12.—Davies Colley's operation, outline of flaps marked out.

They must also be taught to use the muscles of the tongue and palate for the purpose of distinct articulation. Usually there is especial difficulty in pronouncing words containing certain letters, in order to ascertain which, the whole alphabet ought to be repeated and the letters which are difficult to pronounce noted. It will generally be found that *b, d, g, k, l, s, t, z* are pronounced indistinctly. A selection of words containing these letters is made for the purpose of instruction.

The instruction is carried out by a voice trainer, by the mother, or a careful and intelligent nurse in the following manner:—there ought to be nothing to disturb the attention of the child while it is having its lesson. The child should sit facing the instructor, who pronounces the words slowly and in an exaggerated manner with the mouth open and the lips separated. The child then takes a deep inspiration and copies the movements of the instructor's tongue and lips and the sound produced. The words are to be pronounced syllable by syllable. At first the lesson is to be of short duration, care being taken not to tire the child; the periods of instruction are then gradually lengthened.

A useful plan is to mark words difficult of pronunciation in an ordinary



spelling-book—a pictorial one in the case of young children—and make the child practise the slow and deliberate pronunciation of such words.

**OTHER OPERATIONS FOR CLEFT PALATE.**—Space does not permit of a description of Fergusson's and other methods. The method devised by the late Mr. Davies Colley requires notice.

The advantages claimed for the method are that it can be performed at an earlier age. There is less hæmorrhage, no loss of tissue, and very little tension. The disadvantages are that the hard palate alone is united, and a foramen is left at the anterior part of the cleft.

The author strongly recommended his operation in infants, in patients whom the ordinary operation had failed, when the separation of the sides of the cleft is too great to be bridged over in the ordinary way. The soft palate may be closed at the same time or at a subsequent period.

1. A triangular flap consisting of all the soft parts is cut from the side of the hard palate which is the wider, or if the septum be attached to one of the palatal processes the flap is taken from that side. The apex of the flap reaches nearly as far forwards as the incisor teeth, its base is left attached and extends from close to the inner border of the alveolus at the last molar tooth inwards to the edge of the soft palate, near to its anterior attachment. The flap is made by beginning the incision opposite and close to the last molar tooth, carrying it forwards parallel and just internal to the teeth as far as the apex, and then backwards along the edge of the cleft about one-eighth of an inch external to its margin to the line of the base. The flap, along with the periosteum, is then raised by means of a raspatory.

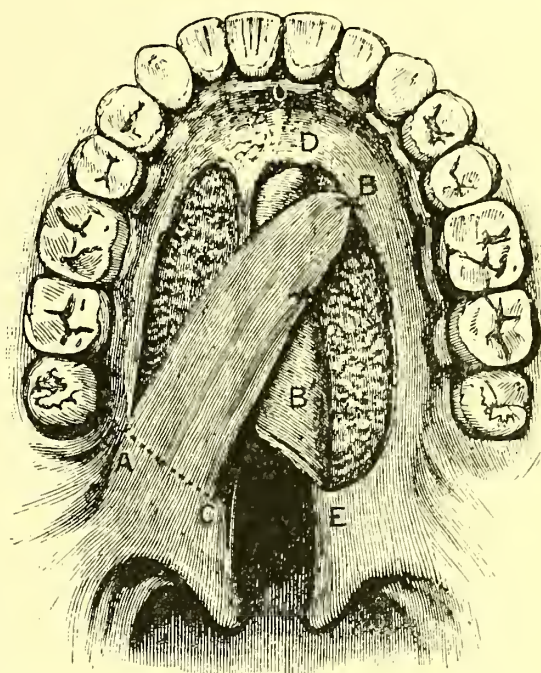


FIG. 13.—Davies Colley's operation, flap A, B, C glided access and united to under raw surface of D, E.

2. A small flap is then marked out on the other side by an incision beginning at the level of the anterior part of the cleft, and carried backwards to the junction of the hard and soft palate at least one-sixth of an inch external to the margin of the cleft. At its anterior and posterior extremities this incision should be carried inwards to the margin of the cleft. A muco-periosteal flap is now detached and turned inwards on its base, which is formed by the tissue at the edge of the cleft, and fixed to the mucous membrane on the opposite side of the cleft by two or three catgut sutures.

3. The large flap is now carried across the cleft, and the anterior parts of its inner margin attached by silver wire or silkworm gut sutures to outer border of the incision upon the other side of the palate.

If there be difficulty in bringing the large flap across the cleft it may be necessary to detach its base more freely from the soft parts which connect it with the hard palate.

The two flaps may be further united by horse-hair sutures. Usually the pressure of the tongue suffices to keep them in position.

**THE MECHANICAL TREATMENT OF CLEFT PALATE.**—As already stated, the treatment of a congenital cleft in the palate by surgical interference is preferable to that by mechanical means.

The obturator, as a rule, cannot be fitted until the habit of defective speech has been fully formed, and as the patient grows frequent renewal is necessary. In certain cases, however, it is the only method of treatment.

In congenital cleft the mechanical treatment is necessary where the gap is very wide and the soft parts are insufficient to close it, where the soft palate is rudimentary, where the os incisivum has been removed and a wide gap exists in front, where operative interference has left a shrunken and deformed palate, and in some cases where the patient has reached adult life.



In acquired conditions mechanical treatment is called for in certain cases of perforation of the palate from traumatism or syphilis, or where a portion of the upper jaw has been removed.

It is unnecessary to enter into a description of these mechanical appliances. They may be obturators or artificial vela, or both, and are to be fitted by a skilled dentist.

The following essential is worthy of attention. An obturator ought to be a plate of vulcanite or gold fitted to the teeth and kept in position by suction. It should never be in the form of a plug, which, by its pressure, causes the opening to become larger.

An artificial velum consists of an obturator with a movable portion attached to it to take the place of the soft palate.

In congenital cases similar instructions as regards articulation have to be given to those already described.

### INJURIES OF THE PALATE

*Mode of Production.*—The palate may be injured in many ways. From very hot liquids, from corrosives or caustics, from fish or meat bones, from a fall with a toy, stick or pipe-stem in the mouth, or from gunshot wounds, usually of suicidal intent.

*Effects.*—These vary according to the nature of the traumatic agent, the part affected, and the degree of sepsis. A hæmatoma may arise, and sometimes be followed by abscess. Sloughing may take place and cicatricial contraction of the soft palate ensue. The hard palate may necrose.

The *soft palate* may be merely punctured or larger wounds may penetrate its entire thickness.

Injuries of the *hard palate*, when extensive, are frequently due to gunshot wounds; in these injuries the nose and pharynx will also be involved. If the injury be not fatal, a large perforation generally results. Necrosis may occur.

*Treatment.*—Foreign bodies may have to be removed. Bleeding, if severe, is to be controlled by ice and pressure. The mouth must be kept as clean as possible by the use of boric lotion, sanitas, or a spray of peroxide of hydrogen, and washed out after food.

Punctured wounds of the soft palate heal without deformity. Large wounds must be sutured.

When the hard palate is involved, any portion of bone and mucous membrane not entirely detached must be retained and the latter fixed in position by sutures.

Gaps in the soft palate and perforations of the hard may have to be dealt with by appropriate measures, such as plastic operations, usually on the principle of Davies Colley's. An obturator is often necessary.

### DISEASES OF THE PALATE

1. *Inflammation.*—*Acute.*—The soft palate may be involved in any of the acute affections which attack the tonsil and pharynx (*q.v.*). Sloughing or suppurative periostitis and necrosis may follow upon injury, or be secondary to dental lesions.

*Abscess of the hard palate* may arise from dental caries and be limited to the anterior part close to the alveolus or pass backwards along the roof of the mouth towards the soft palate. A sinus may result.

*Treatment.*—Early incision of the abscess and attention to the tooth.

2. *Infective Granulomata.*—*Tuberculosis* of the palate is rare as a

primary affection. It is usually associated with similar lesions in the tongue, pharynx, or larynx, or with lupus of the nose and face.

Two forms are met with.

(a) The so-called *lupus* of the palate begins in small miliary growths or nodules which turn yellowish, break down, and becoming confluent leave an ulcerated surface. Between the ulcers, which vary in size, miliary nodules are constantly seen. This association of old and new foci is a characteristic feature of the condition.

The process may extend to the bone, but rarely causes perforation.

(b) The other form is characterised by the formation of large nodules which caseate and then form ulcers. In the hard palate perforation may occur.

Tuberculous periostitis may affect the hard palate, especially in children, and simulate syphilis.

*Diagnosis.*—The difficulty in diagnosis lies between syphilis and tuberculosis when no manifestation other than the palatal lesion is present. The possibility of the two conditions co-existing has also to be borne in mind. Apart from the history and therapeutic test, the appearances of the ulcer and the absence of glandular enlargement in syphilis are of most value.

*Prognosis* depends upon the presence of tubercle elsewhere and upon the tissue or organ affected. In primary tuberculosis of the palate the prognosis is fairly good.

The soft palate may, however, be left cicatricial, deformed, and perhaps adherent to the pharynx; the hard may be perforated.

*Treatment.*—Locally—Lactic acid pure or 75 per cent is perhaps the most satisfactory. Menthol and iodoform are also useful. In some cases the galvano-cautery scraping and excision may be necessary.

Perforation of the soft or hard palate and adhesions of the former may have to be dealt with (*vide* syphilis).

The constitutional treatment is that of other tuberculous affections.

*Syphilis.*—The syphilitic lesions of the palate will not be described in detail (*vide* syphilis).

*Primary* sores are sometimes met with on the hard or soft palate. The diagnosis is often difficult until glandular enlargement manifests itself.

*Secondary Lesions.*—*Erythema* and *mucous patches* and *small ulcers* are common and typical manifestations.

In the secondary period it is not uncommon to find more extensive ulceration, which may destroy a large portion of the soft palate and spread to the hard. The writer has met with several examples of the condition relatively early in the course of syphilis, and usually in elderly and debilitated patients who have not previously been treated constitutionally. The ulceration generally begins near the uvula, and spreads to the soft palate and pillars of the fauces.

The velum may be left deformed, perforated, or adherent to the pharynx.

*Treatment.*—Mercury internally. Locally, peroxide of hydrogen spray and perchloride of mercury. Chromic acid 10 grs. to ʒi. is also useful.

*Tertiary Lesions.*—Under this heading are included the later manifestations of inherited syphilis.

Small firm gumma may be found in the hard or soft palate. In the latter they may arise in the submucosa or muscular substance, in the former in the periosteum, or spread from the nose; this occurs frequently in the inherited form of the disease.

Gumma are usually situated in the middle line, towards the back part of the hard palate and the anterior part of the soft. They are rounded in shape and not infrequently single. The glands are not enlarged.



The course of a gumma varies. Under treatment it may disappear. Frequently it breaks down, or has done so when the patient comes under observation. A gummatous ulcer results. The soft palate becomes more or less destroyed; the hard necrosed and perforated, and the destruction may extend to the bones of the nose, the alveolar border, and to the antrum.

The ravages of tertiary syphilis of the palate sometimes leaves the patient in a deplorable condition. Speech is indistinct, and nasal in twang, food passes into the nose, the bones of which are not infrequently destroyed, and the general health suffers from the concomitant sepsis.

When the process becomes arrested a large gap may exist in the hard palate, the soft be deformed by loss of substance and cicatricial contraction, and perhaps adherent partially or completely to the pharynx.

*Diagnosis* has to be made from tubercle and tumour (*q.v.*).

*Prognosis* is always doubtful. The course of tertiary syphilis of the palate is often rapid, and sometimes extremely difficult to arrest.

*Treatment*.—The patient must be quickly brought under the influence of iodide of potassium, and also, in most cases, of mercury as well. A tonic line of treatment is also usually indicated.

When the gummata have broken down the local treatment consists in combating the sepsis by antiseptic sprays, in employing calomel, and boric powder or starch 1 to 4, and in the use of iodoform.

Necrosed bone is to be removed (*vide* syphilis of bone).

*Treatment of the Results*.—In the soft palate small openings may close of themselves or be closed by operation. Frequently a velum has to be adapted.

If there be adhesion to the pharynx the treatment depends upon the extent of the stenosis. If partial, it may be left alone. If complete, the palate must be detached from the pharyngeal wall, and re-adhesion prevented by the passage of bougies or by an india-rubber ball, which is inflated after it is placed in position.

As regards the *treatment of the perforated palate*. If the opening be large, the patient must wear an obturator; if it be small, an obturator is to be worn for some weeks or months after all traces of the syphilitic manifestations have disappeared, or the patient may plug the opening with gauze, or keep a piece of india-rubber in position over the opening by suction. Operative measures may then be undertaken. Davies Colley's method, or some modification of it, is the most satisfactory.

*Glanders, actinomycosis, and leprosy* affect the palate secondarily.

**TUMOURS**.—*Meningoceles* may be found projecting through the median raphe of the palate.

*Dermoids* are sometimes met with. They may project, according to Bland Sutton, from the buccal or pharyngeal aspect of the soft palate. The tumour is usually composed of connective tissue containing striped muscles and cartilage, sometimes sebaceous material. Skin covers the outside of the mass.

Sometimes small bodies composed of an accumulation of epithelium—the so-called *epithelial pearls*—are seen hanging by short thin pedicles in the mouths of infants. They usually occur in the region of the premaxillæ.

*Cysts*.—Simple mucous cysts, arising in connection with and resembling those met with in the lips, may occur in the palate. Cysts may also arise in the alveolus in connection with the stumps of teeth, and spread to the hard palate. Cystic odontomes may project into the palate.

*Nævi* are occasionally met with, and are best treated by electrolysis.

*Traumatic aneurism* of the posterior palatine artery has been known to arise from a stab and from injury produced by a dental palate.

*Papillomata* occur most frequently on the alveolus. Their most favourite seat is the palatine arches. They present no difficulty either as regards their diagnosis or treatment.

*Lipoma, fibroma*, and other tumours are exceedingly rare.

*Adenomata*.—The tumours hitherto described as adenomata are really *endotheliomata*. They are innocent in character, slow in growth, and encapsuled. At puberty and between 40 and 50 are the ages at which they are most common. They are met with in the soft palate more frequently than in the hard; their common site is at the junction of the two, and are said to be more common on the left side. In shape they are round or oval; as a rule they are inelastic, but sometimes there may be cysts in them. The mucous membrane over them may be normal in character; sometimes it is thinned by pressure of the tumour. In the hard palate they may make a pit in the bone, but they do not grow from it. They give rise to no glandular enlargement. They cause no inconvenience, except from their size, and may be present for some time without the patient being aware of their presence.

There appears to be reason to believe that they may sometimes, after having been present for a long period, suddenly show a rapid increase in growth, ulcerate, and furnish additional evidence of malignancy by causing lymphatic enlargement.

*Treatment*.—Enucleation, in some cases under cocaine, is all that is necessary, unless there be evidence of malignancy, in which case a more radical operation is imperative.

*Sarcoma* may be of the round or spindle-celled variety. Alveolar sarcoma also occurs. These tumours are distinguished from the endotheliomata by their rapidity of growth, by their want of definition, although some have been found to be encapsuled, by the glandular enlargement to which they give rise, and by their tendency to early ulceration. They usually affect the junction of the hard and soft palate.

*Diagnosis*.—From gumma and simple tumours.

*Prognosis* in this class of tumours is grave, and they are often beyond the sphere of successful surgical treatment when they come under observation.

*Treatment*.—Free and early removal of the tumours, together with the glands. The operation is usually one of considerable magnitude, and it may be necessary to perform tracheotomy and ligature the external carotid as adjuvants.

*Malignant Epithelial Tumours*.—This group of tumours may be primary or secondary. When secondary they usually spread to the palate from the tonsil, tongue, or jaws (*q.v.*).

*Primary Malignant Epithelial Tumours*.—Two varieties are met with—epithelioma and glandular cancer.

*Epithelioma* usually affects the posterior part of the hard or the soft palate. It has the usual character of epithelioma, and can as a rule be easily diagnosed. A tertiary syphilitic ulcer of long duration may give rise to difficulty in diagnosis. In such cases a piece of the ulcer should be removed and submitted to microscopic examination. The therapeutic test is only of value if the ulcer *rapidly* improve and heal under antisyphilitic treatment.

*Treatment*.—Free removal of the tumour and the lymphatic glands.

*Glandular Cancer*.—In its early stage glandular cancer, which usually affects the hard palate towards its posterior part, may give rise to considerable difficulty in diagnosis.



The tumour forms a smooth swelling which projects into the mouth, and is usually situated to one side of the middle line. As it grows it spreads out laterally, and also tends to perforate into the nose and antrum. If the case be not seen until the bone has become destroyed there may be considerable difficulty in diagnosing the primary seat of the disease, since, after the bone has become perforated, growth usually takes place rapidly. The buccal aspect of the tumour ulcerates. The lymphatic glands become implicated.

*Diagnosis.*—The gumma is, as a rule, situated in or near the middle line, is round in shape, and does not extend laterally to the same extent as the cancer.

*Prognosis.*—Unsatisfactory, unless an early diagnosis be made and free removal adopted.

*Treatment.*—The removal of the palate process of the superior maxillary or of the antrum, according to the extent of the growth as well as the glands. Some cases are inoperable.

*Paralysis of the Soft Palate.*—*Vide* “Diphtheria,” “Bulbar Paralysis,” “Cerebral Tumours,” etc., etc.

### Congenital Malformations of the Mouth—Hare-Lip

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DEVELOPMENT.—Before entering upon the description of hare-lip, and other allied congenital malformations, a brief account of the development of the face and mouth is necessary.

The object of the writer being to present a summary of the more generally accepted views regarding the malformations to be considered in this article, points of controversy will not be entered into.

About the third week of intrauterine life the primitive cerebral vesicle becomes acutely bent over the end of the notochord, and around the depression thus produced, which soon deepens to form the cavity of the mouth, the face is developed by the blending of various processes.

*Below*, the first pair of branchial arches unite in the fifth week to form the mandibular bar, from which is developed the lower jaw, the lower portion of the cheek, and the chin.

*On each side* a bud-like projection grows in from the upper border of the root of the first branchial arch and forms the maxillary process, from which are derived the whole of the superior maxilla, except the portion which is intermaxillary, the greater part of the cheek, and the labial part of the upper lip.

*From above* a broad process, the fronto-nasal, grows downwards between

the maxillary processes, from which it is separated by a fissure (the naso-orbital fissure).

Thus there are formed around the primitive mouth, or stomodæum, five processes—two mandibular, two maxillary, and the fronto-nasal.

The fronto-nasal process soon, however, manifests a further stage in development by dividing into two lateral and a median process, the latter splitting into two segments, which are separated by a groove.

There are thus two processes on each side—the internal nasal process (globular process of His) and the external nasal process.

From the internal nasal process there develops, superficially, the central portion of the upper lip, deeply, the inner segment (endognathion) of the intermaxillary bone, and above the groove the prominence of the nose.

From the external nasal process is formed the side of the cheek, the ala nasi, and from its deep aspect the outer (mesognathion) of the intermaxilla.

The intermaxillary bone, developed, as mentioned above, from the internal and external projections of the fronto-nasal process, is made up, according to Albrecht, of two triangular portions on each side, known as the endognathion and mesognathion; the maxilla is termed the exognathion. Each segment contains an incisor tooth, and occasionally an accessory tooth is found growing from the endognathion. The apices of the four intermaxillary segments converge towards the anterior palatine canal.

The palate is developed as follows:—About the sixth week there grows inwards from the buccal aspects of each maxillary process a palatal plate, which, by joining with its fellow of the opposite side in the median line, forms the whole of the soft palate and the hard palate, with the exception of the intermaxillary portion.

The hard palate is completed by the junction of the anterior portions of the palatal plates with the intermaxillary portion, a space, the anterior palatine canal, being left in the median line; the entire palatine suture is thus Y-shaped, the junction of the units being at the canal.

The nasal cavity becomes divided into two halves by the growth downwards from the under surface of the fronto-nasal process of a vertical septum which unites with the palate. From this septum the ethmoid, vomer and cartilaginous septum are developed.

The union of the various parts described is completed by the sixth to the tenth week.

The fissure (naso-orbital) which separates the fronto-nasal from the maxillary process is closed, except the part which forms the nasal duct, by the union of the external nasal and internal nasal processes with the maxillary process. The continuity of the upper lip is established by the internal nasal and maxillary processes uniting below the external nasal process.

The external nasal and superior maxillary processes unite first.

The alveolus and upper lip are complete by the ninth week, at which period the union of the palate is commencing from before backwards, being generally completed by the tenth week.

The origin of the congenital malformations of the face and mouth can now be considered from their developmental aspect.

MACROSTOMA arises from non-union of the mandibular and maxillary processes, while MICROSTOMA is caused by the union of these processes passing beyond the normal degree.

MANDIBULAR CLEFT is the result of failure of fusion of the mandibular processes in the middle line.



FACIAL CLEFT is due to non-closure of the naso-orbital fissure.

MEDIAN HARE-LIP presents two forms. The severe form in which, in consequence of the non-development of the internal nasal processes, the central portion of the upper lip, the intermaxilla, and the nasal septum, are absent.

2. In the second variety a median cleft in the upper lip arises in consequence of non-union of the two internal nasal processes. The fissure, if extending to the bones, passes between the two endognathia.

LATERAL HARE-LIP, when simple, *i.e.* limited to the soft parts, arises from failure of union between the internal nasal process with the maxillary process; if alveolar, non-union exists between the same processes superficially, and below and above, and on the deep side between the external and internal nasal processes in addition.

CLEFT PALATE is due to non-closure of the palatal plates. When the fissure passes beyond the anterior palatine canal it may pass between any of the intermaxillary sutures. The cleft usually runs on one or both sides, between the endognathion and mesognathion.

CONGENITAL DEFORMITIES OF THE FACE AND MOUTH.—The rarer conditions will be briefly referred to, and the commoner forms, hare-lip and cleft palate, considered in greater detail.

MACROSTOMA.—Non-union of the maxillary and mandibular processes give rise to this deformity, which consists in enlargement of the mouth in the transverse diameter. The extent of the fissure varies from a slight enlargement of the angle of the mouth to a gap in the cheek reaching to the last molar tooth, and sometimes as far as the auricle.

If unilateral, the deficiency is usually on the right side; not infrequently the condition is bilateral. There may be other associated deformities, such as accessory auricular appendages, defective formation of the external ear, hare-lip, cleft palate, an imperfectly developed brain.

*Treatment.*—Operative treatment is necessary, not only to remedy the deformity, but to improve the nutrition of the child. When the cleft is extensive suckling is difficult, if not impossible, food is not retained in the mouth, and saliva constantly escapes.

The edges of the cleft must be pared and carefully united by suture, attention being specially directed to the mucous membrane at the angle of the mouth.

MICROSTOMA is a congenital narrowing of the mouth, brought about by union taking place between the mandibular and maxillary processes to an abnormal extent.

In severe cases the opening representing the mouth may be scarcely large enough to admit a probe; not infrequently there is a deficiency of the lower jaw.

*Treatment.*—In well-marked cases a mouth is to be formed by making a transverse incision on either side, and accurately uniting the mucous membrane to the skin.

MANDIBULAR CLEFT is one of the rarest of the congenital deformities met with in connection with the mouth. Its etiology lies in the non-closure of the lateral segments of the mandibular processes.

The degree of deformity varies to a considerable extent. Complete closure of the processes may be represented merely by two sinuses or openings in the mucous membrane of the lower lip. Such sinuses discharge mucus secreted by the glands which line their walls, and are not uncommonly associated with double hare-lip. A similar sinus has been met with in the upper lip, occupying the site of an ordinary hare-lip.

True mandibular cleft may involve the soft parts only, or extend to the bones. Occasionally the defect may be associated with a dermoid; in some cases the tongue has been found to be cleft, in others the face.

*Treatment.*—Pare the edges and close the cleft.

FACIAL CLEFT is an exceedingly rare malformation, due to non-closure of the fissure between nasal and maxillary processes.

The cleft, which begins in the upper lip, usually at about the same place as that met with in ordinary hare-lip, passes upwards and outwards external to the ala nasi towards the centre of the lower lid or to the inner canthus; sometimes it extends across the orbit into the temporal region.

The deformity varies in extent. As a rule, the soft parts alone are involved. When the bones are implicated, the deficiency in the alveolus is situated, as in hare-lip, between the endognathion and mesognathion; there may also be a large opening into the antrum.

The cleft may be unilateral or bilateral. It may be partially or entirely represented by a thin cicatrix. Macrostoma, mandibular cleft, and coloboma of the iris or the choroid, have been found as associated conditions.

*Treatment.*—Closure of the cleft by suitable plastic operations.

MEDIAN HARE-LIP.—Two varieties of these rare conditions are described.

1. There is a wide median gap in the upper lip, due to the entire absence of its central portions, with, in addition, absence of the intermaxillary bones and of the nasal septum.

The nose is much flattened, and there may or may not be a cleft in the palate.

*Non-development* of the internal nasal (globular) processes accounts for the deformity.

2. In a second variety a median cleft, of varying extent, is present in the upper lip, due to *non-union* of the globular processes. There may be no other deformity, but in some instances the intermaxillæ have been found to be separate, the palate cleft, the anterior nares separate, and the nose flat.

*Treatment.*—Operative methods on the principles of those employed for ordinary hare-lip and cleft palate.

LATERAL HARE-LIP presents many varieties. It may be *unilateral* or *bilateral*. Its extent may vary from a slight notch at the margin of the lip, or it may pass into the nostril—*simple hare-lip*. The alveolar margin may be involved—*alveolar hare-lip*. Or a cleft of the palate may be associated with the labial deformity—*complicated hare-lip*.

UNILATERAL HARE-LIP.—The left side is usually affected. The cleft is  $\Lambda$ -shaped, with rounded margins; the red border passes upwards for a varying extent; in some cases skin forms the apex of the cleft. The angle at the nose may be acute or obtuse, the two sides of the fissure equal in length and inclination; frequently they are unequal.

In consequence of the continuity of the orbicularis being interrupted by the fissure, and of the contraction of the muscles at the angle of the mouth, the deformity becomes aggravated with age, and best marked when the child cries. This occurs notwithstanding the fact that the lip is frequently bound down to the alveolus by reflections of mucous membrane. The frenum is frequently long.

*The nostril* is often flattened, even in cases where the cleft does not extend into the nose. When the fissure passes into the anterior nares, the flattening is more extensive, and it becomes extreme where the posterior wall and floor of the anterior nares are deficient.



**ALVEOLAR HARE-LIP.**—The bone may be partially or completely cleft, and the fissure may extend backwards to the palate. The floor of the nostril is not always implicated. According to Albrecht, whose views meet with most acceptance, the site of the alveolar cleft is between the endo- and mesognathion. Kölliker maintains the cleft to be mesognathic, *i.e.* between the intermaxillary bone and the maxilla.

The edges of the alveolar cleft may be on the same level; frequently they are unequal. If a complete cleft palate be present, the fissure extends backwards and inwards to the anterior palatine canal, and thence backwards to the uvula.

**DOUBLE HARE-LIP.**—When simple, the two fissures are often unequal in extent; thus the nostril may be involved on one side and not on the other. The outer segments of the cleft have the same characters as that met with in cases of unilateral cleft.

The central segment (prolabium) is generally smaller than normal, and frequently attached to the bone beneath (os incisivum), which usually protrudes beyond it.

There may be a single or double alveolar cleft.

In cases of double alveolar cleft, the os incisivum, formed by the united endognathion (containing in the young child the rudiments of two pairs, temporary and permanent incisions), may remain in its normal position; usually it is pushed forward and rotated, so that the alveolar margin projects in front. Cleft palate is present, and the total cleft is  $\Lambda$ -shaped.

In aggravated cases the os incisivum, together with the adherent prolabium and shortened columna, supported by the vomer and nasal septum, form a proboscis-like appendage to the end of the nose.

**THE EFFECTS OF HARE-LIP.**—When the lip alone is involved, and only to a slight extent, the general health does not suffer, and the infant can take food in the natural manner. In the severer forms the patients are frequently feeble and badly nourished. This condition of malnutrition is not due entirely to difficulty in feeding. Such infants are weakly and feebly developed, and sometimes die in spite of the careful administration of food. Exposure of the mouth and naso-pharynx in these complicated cases render attacks of catarrh of common occurrence.

**TREATMENT OF HARE-LIP.**—Hare-lip can only be cured by operation, the objects of which is to close the cleft as early as possible, to avoid risk to life and to obtain a satisfactory result.

The attainment of these ends falls to be considered under age, state of health, and essentials for success in operating.

1. *Age.*—The simpler the case the earlier can the operation be undertaken. In the severer forms there is a greater loss of blood and more shock, and surgical treatment is often better delayed. The operation ought to be performed before the sixth month, that is before primary dentition begins, unless there be some special reason for delay. It may be stated generally that the best time is between the sixth and twelfth week. At this period the child is better able to stand the loss of blood than at an earlier date, and the deformity has not become aggravated by delay.

Some surgeons operate during the first few days or weeks of life, and this may be done in strong healthy children and in slight cases.

When cleft-palate complicates hare-lip the latter should always be dealt with first in the case of young children. The operation on the lip improves the palate condition. In children, after the fourth year, it is more convenient to treat the palate first and the lip subsequently.

Those who operate upon the palate in infants also follow this order in operating.

2. *The state of the child's health.*—The health of the patient should be in the best possible condition.

Feeble infants stand surgical interference badly, and a relatively severe operation may be attended by risk to life; further, loss of blood and its attendant shock predisposes towards sepsis, which is an important factor in the production of failure of primary union.

If a feeble state of the general health arise from inability to take a sufficient quantity of food, careful feeding must be employed from birth. In some cases a feeding-bottle with an extra large teat will meet the requirements, in others a spoon must be employed. The head should be thrown well back, and the milk dropped into the back of the pharynx. Badly-fed and half-starved children speedily improve as a rule under this line of treatment.

There remains, however, a group of cases in which the careful administration of food does not effect an improvement; in such, the risks attendant upon operation must be carefully considered before it is undertaken.

Hare-lip operations should not be performed during the period of dentition, while the presence of coryza, stomatitis, bronchial or gastrointestinal catarrh, or congenital syphilis call for delay. It is a good rule to leave the child under observation for some days prior to operating.

3. *Essentials for success in operating.*—In all operations for hare-lip certain essentials have to be borne in mind, of which the following are the more important:—

Healing should take place by primary union. This is to be attained by asepsis, and by the absence of all tension.

The flattening of the nostril must be overcome by freely separating the adherent lip from the alveolus.

The separation is best effected mainly, if not entirely, on the outer side of the cleft. By adopting this plan the central portion acts as a fixed point and helps to maintain the nostril in position. Sometimes the separation may have to be carried up to the infra-orbital foramen. The ala of the nose may have to be freed, and in bad cases it may be necessary to carry an incision around the nostril. In effecting the separation the knife or scissors must be kept close to the bone, and bleeding arrested by pressure.

A satisfactory cicatrix must result from the operation. The edges of the cleft must be freely pared, the raw surface being as wide as possible, skin accurately sutured to skin, and mucous membrane to mucous membrane, so as to leave the red margin continuous; tension is to be avoided by free separation of the lip (*vide supra*). Faulty paring and careless suturing leave a thin and uneven cicatrix, which is prone to gradually stretch.

A notch at the red margin is to be avoided by paring the edges in a concave manner, or by turning down or across flaps or a flap (*vide Operations*). The resulting projection (prolabium) ought to be rather excessive to allow for subsequent contraction.

The additional essentials for complicated cases are described under the various operations.

*Methods of Operating.*—Space does not permit of a description of the many operations which have been practised for the relief of hare-lip. Only those which the writer has found to be most satisfactory and to meet the requirements of most cases will be described.



The various procedures may be considered under the headings of those employed in cases of single and of double hare-lip and their varieties.

*The Instruments required.*—A sharp and narrow-bladed knife, two pairs of catch forceps, blunt-pointed scissors, artery forceps, periosteum elevators, strong forceps such as sequester forceps, with the blades covered by pieces of drainage-tubing, bone forceps, and a fine saw.

Fine sponges, sponge-holders, sterile lint or gauze.

Fine cat-gut for ligatures. For sutures, silver wire of medium thickness, silk-worm gut, horse-hair, and possibly cat-gut. Hare-lip pins are seldom necessary, and are not to be recommended.

Collodion, and strips of gauze cut in the shape of a dumb-bell for dressings.

*Steps of the Operation.*—The child should have its head slightly raised and steadied by the anæsthetist, and the movements of its arms controlled.

Chloroform is the more suitable anæsthetic.

The surgeon stands on the side to be operated upon, or at the head of the patient. His assistant controls the coronary arteries by grasping the lip between the finger and thumb, and sees that no blood enters the air passages.

OPERATIONS FOR UNILATERAL HARE-LIP.—I. When the cleft is partial, and the nose not flattened.

The simplest operation is Nélaton's. A  $\Lambda$ -shaped incision is made parallel to, and just outside, the edge of the notch through the whole thickness of the lip. The mucous membrane is drawn down, and the  $\Lambda$  incision converted into a  $\nabla$ . Sutures are now inserted, and the  $\nabla$ -shaped opening closed, so that a vertical cicatrix will result, with a projection of the red margin of the lip. The projection may at first be excessive, but it afterwards diminishes in size. The stages of the operation may be represented thus  $\Lambda$ ,  $\nabla$ .

This method is not so satisfactory as a primary operation, but is extremely useful when, as the result of a previous operation, a notch has been left at the red margin of the lip.

A better plan is to pare the edges so as to leave a concave surface on each side, at the same time removing the apex of the cleft, or to employ one of the methods advised for a complete cleft.

II. In partial cases, where the nose is flattened, a strip of the whole thickness of the lip must be removed by an incision prolonged into the nostril, and the ala nasi freed, as in cases of complete cleft.

III. When the cleft is complete, and extends into the nostril.

1. The lip and ala nasi must be freely separated from the bone in the manner already described.

2. The edges are pared.

Many methods have been employed, which vary according to the degree of symmetry of the margins of the cleft.

(a) The simplest method, where the margins are equal, is to pare them by carrying incisions directly downwards, and parallel to the outside edge of each cleft, and to prolong the incisions sufficiently to remove the rounded margins of the cleft at the red border of the lip. This method, which is frequently employed by Mr. Annandale, yields excellent results.

(b) The incisions may be made parallel to the margins of the cleft, terminating just above the labial margins, at which spot two transverse incisions are made in the substance of the lip. This enables two tags or flaps to be turned down, in order to form a projection. If the tags be too long, they may be shortened to the necessary extent.

(c) The method of Rose yields very satisfactory results.

The edges of the cleft are pared in a crescentic manner until the muco-cutaneous junction is reached; the knife is then turned, and the mucous membrane of the lip cut at an angle of about  $60^\circ$  to the former incision. The depth of the lip is thus increased, and no notch results.

If this method be employed where the nose is much flattened, more tissue ought to be removed from the outer side than the inner.



Where there is a wide gap, and marked inequality of the margins of the cleft, the flap method of Mirault, or one of its numerous modifications, is very serviceable.

The inner mesial margin is pared in an angular method, so as to leave a raw surface; the outer margin is pared, and a flap as thick as possible obtained from the outer side. The edges of the pared cleft are then united, and the flap brought across and attached to the inner margin.

When the nose is much flattened the outer wall of the nostril should always be rawed, and also the opposing side of the septum, so that these surfaces may be brought together.

Instead of paring the edges of the cleft, they may be split. This method, which saves sacrifice of tissue, is especially useful where the margins of the cleft are thin. It is founded upon the principles of the operation, introduced by the late Mr. Duncan many years ago, for the closure of fæcal fistula, and has been strongly advocated by Mr. Chiene.

The lip is split at the junction of skin and mucous membrane nearly down to the red margin, sufficient room being left to turn down two tags to form a prolabium. The fissure is closed by uniting the skin to skin, and mucous membrane to mucous membrane.

*Unilateral Alveolar Hare-Lip.*—In this variety the deformity is usually great, and the nose markedly broadened. The margins of the cleft alveolus may be on the same level; usually they are not. A useful preliminary to the operation, in cases where the margins are unequal, is to have gradual pressure made by the mother or nurse upon the projecting margin, combined with outward traction upon the depressed side. By this means considerable improvement is often brought about. If this has not been done, the pressure of the sutured lip will suffice in slight cases to overcome the projection.

When the deformity is very marked the projecting margin of the bony cleft must be forced into position by strong forceps, the blades of which are covered by rubber tubing, or it may be necessary to fracture the prominent alveolus or divide it with a fine saw about one inch external to the cleft, and, having pared the edges of the cleft, to suture them into position.

In order to overcome the extreme broadening of the nostril an incision curved round the ala nasi may be required.

The other steps of the operation are similar to those already described.

*Double Hare-Lip.*—The operation for the cure of complicated cases of double hare-lip will be considered under the headings of (1) simple cases, where the os incisivum retains its normal position; (2) where the bone projects forwards.

1. Where there is no projection—

(1) The soft parts are freely separated from the jaws, and the prolabium from the os incisivum.

(2) The margins of the cleft are pared by one of the methods already mentioned. The prolabium is also pared in a rectangular or V-shaped manner, so that it will fit in between the pared edges of the lip when they are brought together.

When the cleft is complete the incision must be carried into the nostril in the manner already described, in order to overcome the flattening of the nose; and in aggravated cases it is often necessary to carry an incision around each ala nasi, and to freely separate the nostril from the bone. In these cases the removal of an elliptical portion of skin allows the lip to come readily together without giving rise to undue narrowing of the nose.

The precautions already mentioned as necessary to prevent a notch at the red margin must be adopted.

The method of closing the pared cleft merits attention. The prolabium must not be brought down to the red margin, otherwise the tip of the nose will be depressed. In cases of moderate severity the apex of the V is fixed above the middle of the new lip. In others the prolabium can only be used to form a columella, and merely its tip brought between the lateral segments.

2. Where the os incisivum projects, the methods of dealing with the projection vary according to the degree of deformity. The bone may be replaced, excised subperiosteally, or entirely removed. Complete excision leaves the central portion



of the lip unsupported, and ought to be reserved for cases where the projection is very great or its neck of attachment very narrow.

(1) The soft parts are freely separated from the bone, and the projection is forced into position and the cleft closed.

(2) In greater degrees of deformity the operation is best done in two stages—the central portion dissected off the bone, which is dealt with by one of the following methods, and a week or ten days later the cleft is closed.

(a) *Λ-shaped portion* may be removed after the mucous membrane and periosteum have been detached from the septum. The bone is then replaced, and retained in position by wire sutures passed through the alveolar margins. If the sutures be left somewhat long the child's tongue will not press against the bone.

(b) *Partial removal* of the bone overcomes the deformity, and the subsequent formation of new bone supports the upper lip. An incision may be made along the lower border of the projection, and a portion of the bone together with the teeth be gouged away, or, a subperiosteal excision may be performed through a similar incision, and the remaining flaps united to the anterior and posterior margins of the rawed alveolar borders.

(c) *Entire removal* of the os incisivum is accomplished by dividing the neck with bone forceps. Bleeding can usually be arrested by pressure; sometimes a touch with the cautery is necessary.

Closure of the cleft is effected in the manner already described. The broadened nostrils and shortened columella require especial attention.

In aggravated cases the closure of the cleft may be performed in two stages, one side being operated upon first and the other subsequently closed. This method in cases of extreme deformity yields excellent results.

*Suturing the Cleft.*—The best materials are silver-wire or silk-worm gut for the deep, and horse-hair for the superficial stitches. Catgut is employed by many for the mucous membrane. Hare-lip pins are seldom advisable or necessary.

The cutaneous portion of the cleft should be carefully sutured, care being taken to prevent inversion of the edges, and the red margins of the lip, the projecting tags of mucous membrane, or the flap, according to the method of paring which has been employed, drawn evenly and accurately together.

The number of deep sutures necessary (silver-wire of moderate size is the best material) when the gap is a wide one will depend upon the extent of the cleft. The needle is introduced about one-third of an inch from the rawed surface and carried down to, but not through, the mucous membrane. If this plan be adopted it is not necessary to unite the mucous membrane on the buccal aspect by catgut. In a complete cleft three deep stitches are usually required—one at the top of the cleft close up to the nose, a second about the middle, and the third just above the red margin of the lip. All these sutures are passed, and the accuracy of their position tested before the wire is tied. Two or more intermediate superficial sutures of horse-hair are then introduced, and finally the red margin is brought accurately together on its external, internal, and inferior aspects by very fine horse-hair, or by catgut, if that material be preferred.

In Mirault's operation or its modifications the first deep suture is passed so as to draw the angle of the pared inner margin into that formed by the flap turned down from the outer margin.

In double hare-lip the upper wire suture should pass through the lateral portions and the base of the central V-shaped portion, if that be of sufficient size, the second through the apex of the V, while the third unites the lateral portions only. Where, however, the columella is short the upper suture passes through the apex of the V.

*Dressing.*—The parts are carefully cleansed of blood, and in many cases a dressing is not necessary. The wound can, however, be protected and steadied by applying a dressing of sterile gauze cut in the shape of a dumb-bell. One of the ends is fixed by means of flexile collodion; when it is dry the cheeks are pushed forwards, the narrow portion carried across the wound, and the other end fixed to the cheek by collodion. The cheeks are held forward until the collodion is dry. No collodion is applied to the wound.

After the stitches have been removed, plaster cut in a similar manner is used for three or four weeks in order to prevent the cicatrix stretching.

**AFTER-TREATMENT.**—Where the gap has been wide and the nostrils much flattened, the child has to be carefully watched for at least twenty-four hours after the operation, lest it suffocate. A small piece of tubing



may be passed into the nostrils, and the nurse should be instructed to see that the lower lip does not act as a valve, and to draw it down with the finger from time to time.

Care should also be taken to prevent the child pulling at the sutures.

The patient should be carefully and regularly fed with a spoon. No food should be allowed to touch the lip until the wound is healed.

The stitches can, as a rule, all be removed by the end of a week. The wire sutures may be taken out, or one or more of them divided and left in position from the fourth day onwards. The horse-hair sutures are the last to be removed.

COMPLICATIONS.—If primary union completely fail, and the child's health be satisfactory, the edges of the wound may be at once rawed and brought together; if failure be partial, carefully drawing the edges together with plaster will usually bring about union by granulation. The cicatrix will, however, be more or less a broad one, and the lip will have to be supported for a considerable time in order to prevent stretching.

Death sometimes occurs after the operation in feeble children, from lung or other complications.

Faulty results may have to be remedied. A notch in the red margin may be treated by Nélaton's or other methods. The flattened upper lip met with after the removal of the os incisivum can be improved by the adjuvant of a plate with artificial incisions.

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### **Palmar Fascia.** See DEFORMITIES, FASCIA.

**Palpitations** is the too forcible or too frequent beating of the heart, whereby its action becomes perceptible to the individual. It is due to an increased excitability of the heart muscle, and may be induced by a variety of different causes. It is an unimportant symptom in the majority of cases of organic cardiac disease (see vol. iv. p. 383), and is usually dependent on a so-called functional disorder of the heart. The subjective symptoms associated with it vary in degree and kind according to the individuality of the patient. Some subjects merely experience a slight discomfort from the sensation caused by the unnatural impact of the heart; in others the distress is considerable. Objectively the apex beat is, as a rule, too diffuse and sharp in character, with irregularity in action. In severe cases, the whole precordia may be the seat of impact, more especially in those cases where the symptom is one of organic cardiac disease. The degree of subjective disturbance bears no constant ratio to the objective change. The causes are predisposing and exciting. Of the former, a naturally nervous constitution is by far the most important. The exciting cause is usually to be found in a temporary derangement of one or other viscus, and more especially those rich in nervous connections with the central nervous systems *via* the sympathetic nervous system.



Gastric and intestinal derangements probably account for more than nine-tenths of the cases. Constipation and a faulty dietary are responsible for these. Some degree of hepatic derangement is usually associated with and dependent on this gastro-intestinal disturbance. The most striking cases that come under observation are probably those which have been at an earlier date the subject of dilatation of the heart, and which have made a very slow, and it may be an incomplete, recovery. If this occurs in a nervous subject, we have the ideal ground for the frequent development of those subjective sensations that are popularly called palpitations, and a slight derangement of the digestion may readily induce the symptoms. With regard to treatment, the first essential is to relieve the patient's mind as to the relatively unimportant nature of his ailment. In the second place, we have to look for a source of reflex irritation in the gastro-intestinal canal and its adnexa and other organs, and counteract it. A blue pill, followed by a morning saline and a simple diet, constitutes the best line of treatment. Constitutional remedies are sometimes called for, and then a short rest from active physical or mental work, with gentle exercise in the fresh air and a general tonic, will be found most useful. Local treatment—the applications of a Belladonna plaster, or in chronic cases a cantharidis blister—is serviceable in some cases, and in this class of cases the administration of bromides and other cardiac sedatives will conduce to a more rapid recovery. The practitioner must judge for himself the state of the cardiac muscle. A careful investigation of the case may suggest the advisability of administering small doses of a direct cardiac tonic, *e.g.* digitalis, the use of which may be attended with marked benefit. But great care is called for in the selection of cases suitable for this line of treatment.

Pancreas.

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THE pancreas is a compound tubular or tubulo-acinar gland. The main duct or duct of Wirsung and its larger branches are lined by columnar epithelium, which is 13 to 18  $\mu$  high in the large divisions, and becomes lower (5 to 7  $\mu$ ) in the smaller or interlobular branches. External to the epithelium lies elastic fibrous tissue, containing blood-vessels, lymphatics, and nerves. The latter is continued into the glandular substance of the pancreas, subdividing it into lobes and lobules. The interlobular ducts end in "intermediate pieces" or ductules lined by long (12  $\mu$ ), flattened (about 2 to 3 m. in height) epithelial cells. These ductules subdivide repeatedly within the lobule before they terminate in the secretory or glandular subules. At their junction with the secretory tubules their lining epithelium is in some parts continued a longer or shorter distance into the interior of



the secretory tubules, thus forming an epithelial layer internal to the glandular epithelium proper. In consequence of their position they have been described by Langerhans as the centro-acinar cells. In stained preparations they may be readily distinguished from the true secretory cells by their shape and the characters of their nuclei, which are ellipsoidal and poor in chromatin.

The true glandular tissue of the pancreas, in which the duct system terminates, consists of long, highly convoluted and branched tubules, possessing numerous lateral diverticula. The lumen of the tubules, which vary from 1 to 2  $\mu$  in diameter, is bounded by cells having a more or less cone-like form, the blunt apex of the cone being directed towards the lumen. The form of the cells, however, varies considerably, so as to become adapted to the manifold convolutions and diverticula of the tubules. In their greatest diameter they vary in size from 7 to 19  $\mu$ . Microscopical examination of the pancreas of the living rabbit shows that the protoplasm of each cell consists of two zones—an inner granular and an outer, more homogeneous zone, showing a faint longitudinal striation. During secretion the granules of the inner zone gradually undergo solution and extrusion into the lumen of the tubule. The tubules of the actively secreting glands may readily be distinguished from those of the resting gland by the fact that the basement membrane is thrown into folds corresponding in position to the subjacent cells. The outlines of the cells are sharply marked off in the actively secreting tubule, whereas in the resting tubule they are indistinct and the contour of the basement membrane is smooth.

In preparations fixed with alcohol and stained with carmine, the outer zone is stained of a bright red, while the inner zone is only faintly stained. During hunger the granular inner zone is broad, the finely striated outer zone forming the narrow external border of the cell. During the first period of digestion the inner zone diminishes in size, the outer zone increases, and the cell, as a whole, becomes slightly smaller. After active secretion has ceased, a gradual restitution of the cell to its condition during hunger is established. Changes similar to those during the first stage of digestion may also be produced by the injection of pilocarpine. Heidenhain has shown that the quantity of trypsin present in the secretion varies directly with the development of the inner granular zone. In the case of dogs, in which a permanent pancreatic fistula had been formed, he found that the greatly diminished activity of the pancreatic juice was associated with almost entire absence of granules from the inner zone. He has shown that trypsin is only present in the pancreatic juice, not in the resting cells, since a fresh pancreas in the resting condition, showing a well-developed granular zone, yields on extraction with glycerine little or no trypsin, but its precursor, or zymogen. He therefore concludes that the granules within the cells contain only zymogens. The zymogen granules are highly refractile, easily soluble in alkalies, and become brown when treated with osmic acid. These facts induced early observers to regard them as fatty globules. They are, however, much less stable than fat rapidly swelling and then undergoing solution when treated with water. On account of this instability the granules are difficult to fix, the deeper parts of the tissue often escaping the action of the fixatives, the best of which for this purpose are corrosive sublimate and osmic acid. The granules lie in the interstices of a spongioplasmic meshwork, which stains readily with acid dyes. In the outer zone of the cell the spongioplasmic strands are arranged longitudinally, and appear to be amphophil in their relation to staining reagents. At the junction between the two zones lies the nucleus, which in the living cell is spherical;



while in fixed preparations it is often ellipsoidal and slightly shrunken. When suitably stained, it shows a somewhat wide-meshed chromatic network, and one or occasionally two nuclei.

By means of injection and the use of Golgi's method, it has been shown that intercellular secretory capillaries extend laterally from the central lumen of each glandular tubule between the cells as far as the outer limit of the inner granular layer. None of these capillaries reach the basement membrane. The secretory capillaries appear to end within the cells in secretory vacuoles. The latter, which may be readily demonstrated by the use of Golgi's method, are not to be regarded as fixed preformed structures—since they vary much in number, size, and position—but as resulting from the solution of zymogen granules during secretion.

Scattered throughout the substance of the pancreas, usually in the interior of the primary tubules, more rarely at the periphery, occur a number of rounded or irregularly-formed masses of cells, ranging in diameter from .07 to .3 mm. These groups of cells have received the name of Langerhans' cell islets, and are present in the pancreas of all those mammals in which they have been looked for, as well as in the pancreas of birds, reptiles, and amphibians. They are separated from the rest of the gland substance by a more or less distinct connective-tissue capsule. The epithelial cells within the islet are arranged as a meshwork of anastomosing cell columns. The spaces left between the cell columns contain large capillary blood-vessels whose endothelial walls lie in direct contact with the epithelial cells. As a rule, each cell column consists of two or more rows of cells. Occasionally, however, a cell column may be found consisting of a single row of cells. Ducts, although sought for by numerous observers, have not been discovered, so that the only channels by which the products of activity of the cells can be transported are the blood and lymph streams. The rich supply of capillary blood-vessels, and their intimate relation with the epithelium, obviously favour metabolic interchange between the epithelium and the blood.

The epithelial cells, forming an islet, may readily be distinguished from the glandular cells of the pancreas proper by the absence of the division into an inner granular and an outer, more homogeneous zone, and by the uniformly faintly granular character of their protoplasm. The cells, which vary from 9 to 12  $\mu$  in diameter, are roughly polygonal in shape, and in fixed preparations are seen to possess a finely meshed spongioplasm. Their nuclei are ellipsoidal, have a fine chromatic network, and contain nucleoli of much smaller size than those present in the nuclei of the glandular cells of the pancreas proper. Occasionally a nucleus of unusually large size may be found.

The pancreas of the human subject develops as a single dorsal protrusion and a double ventral protrusion of the hypoblast lining the upper part of the small intestine. The ventral protrusions later on fuse to form a single mass, and at a still later date fusion of the dorsal and ventral precursors of the gland takes place. The original duct of the dorsal protrusion becomes the duct of Santorini, while the duct of the ventral part becomes the duct of Wirsung. Laguesse has shown that, in the case of the sheep, the cell islets of Langerhans develop from the same region of the hypoblast as the pancreas proper.

While the pancreas has long been known to produce an external secretion, it has only recently been shown to exert an important influence on general metabolism. For fuller details with regard to the nature, mechanism of production, and nervous control of the external secretion,



reference must be made to the article on "Digestion and Metabolism." A *résumé* of the main facts with regard to the chemistry of the gland and its secretions, together with a more complete account of its influence on general metabolism, forms the contents of the remainder of this article.

The pancreas of the human subject contains about 74·53 per cent water, 24·57 per cent of organic, and 0·95 per cent of inorganic solids. The chief organic solids are albumin, globulin, nucleoproteid, and the enzymes found also in the external secretion. Leucin, tyrosin, and the purin bases—xanthin, hypoxanthin, guanin, and adenin—are the chief nitrogenous extractives. Lactic acid, volatile fatty acids, neutral fats, and inosite, are the chief non-nitrogenous extractives. The most characteristic proteid constituent, the nucleoproteid  $\alpha$ , obtained by Hammarsten from the pancreas of the ox, is soluble in water and dilute alkalies, precipitated from its solution by dilute acetic acid, and possesses a powerful proteolytic action. On heating its solution, nucleoproteid  $\alpha$  splits up into a coagulated albumin and a nucleoproteid  $\beta$ , containing 4·48 per cent phosphorus. The latter is soluble in water and dilute alkalies, and precipitable by the addition of dilute acetic acid. On digestion with pepsin in acid solution nucleoproteid  $\beta$  yields, amongst other products, a nuclein from which Bang prepared a nucleic acid—guanylic acid—with marked differences from the other known nucleic acids. It is soluble in water and dilute alkalies, and precipitable by dilute acetic acid as well as by dilute mineral acids. While the normal relation of nitrogen to phosphorus in nucleic acid is 3:1, in guanylic acid the relation is 5:1. The nucleic acid prepared from the nucleoproteid of the pancreas has received the name guanylic acid because only one purin base, guanin, has as yet been obtained from it. Amongst the products of its decomposition is a pentose which appears sometimes to be present in the urine in cases of pancreatic disease, and whose presence there may in the future be useful as an aid to differential diagnosis. In 64 out of 80 cases of diabetes in the human subject, Külz found a pentose present in the urine. The results of Jolles indicate that a nucleoproteid yielding pentose on heating with dilute acids occurs in the urine in some cases of pancreatic disease. After saturating with magnesium sulphate a urine from which he had been previously able to prepare an osazone, Jolles found that the filtrate yielded no osazone; while the precipitate, which contained phosphorus in organic combination, after being heated with a dilute mineral acid, yielded a solution from which a pentosazone could be prepared.

The external secretion of the pancreas may be best obtained in a pure state from an animal having a permanent fistula made by Pawlow's method. It is a clear, colourless, odourless, and alkaline fluid, containing, along with coagulable proteid, four enzymes, a proteolytic, trypsin, a fat-splitting steapsin, an amylolytic, amylopsin, and a milk-curdling ferment. In addition to the above, it contains leucin, tyrosin, and small quantities of fats and soaps. The chief mineral constituents are chlorides of the alkalies, especially sodium chloride, alkaline carbonates and phosphates, as well as small quantities of calcium, magnesium, and iron. The composition of the juice varies considerably, according to the nature of the diet. Any variation in the relative proportions of proteid, carbohydrate, and fat in the diet is followed by a corresponding variation in the relative proportions of the enzymes necessary for their digestion.

Ligature or obstruction of the pancreatic duct leads to a diminution of the digestion and absorption of the various food-stuffs without any direct influence on general metabolism; while complete excision of the gland produces, in addition to these effects on digestion and absorption, the condition



known as pancreatic diabetes. With regard to the effects of removal of the pancreatic juice from the intestine on the digestion of proteids, carbohydrates, and fats, the results of different observers vary considerably; 18 to 80 per cent of the proteids of the food, and about 50 per cent of the carbohydrate, when given as polysaccharides of glucose, are stated to be absorbed. Fat, except in the form of the fat in milk, is not absorbed. Harley states that dogs are in some cases only able to absorb 4 per cent of the fat of milk, while Minkowski found that 28 to 53 per cent was absorbed. Probably the difference in the observations of Harley and Minkowski is due, in part at least, to variations in the extent of bacterial decomposition occurring in the intestines of the dogs examined.

After complete excision of the pancreas in dogs a diabetes of the severest type invariably follows: Extirpation of the pancreas produces the same effect on cats. In rabbits, extirpation is rendered almost impossible by the anatomical relations of the gland, and experiments in this direction have led to doubtful results. Minkowski succeeded in almost completely excising the pancreas of a pig, and found that a diabetes of medium severity followed. So far as researches on birds have been carried out, it appears that in birds of prey diabetes follows excision of the pancreas, whereas in geese and pigeons Minkowski's experiments led to negative results. More recently Kausch has succeeded in producing diabetes in geese and ducks by excision of the pancreas. After excision of the pancreas in frogs no sugar appears in the urine until some time—usually five days—after the operation. The following description refers mainly to the effects produced by extirpation of the pancreas in dogs.

The excretion of sugar does not commence immediately after the operation. In the majority of cases only traces up to 1 per cent are found in the urine during the first twenty-four hours, on the following day about 4 to 6 per cent, and only on the third day does the excretion attain a maximum of about 8 to 10 per cent. If at this stage no nourishment be given, the quantity of sugar excreted gradually sinks; yet, even after the dog has remained without food for seven days, sugar is still to be found in the urine. The quantity of sugar excreted can necessarily only be a measure of the severity of the diabetes if carbohydrates be excluded from the diet, or if the quantity of carbohydrates present in the food and faeces be also estimated. If the animal be kept in a state of hunger or on a purely proteid diet, the quantity of sugar excreted, however variable from day to day, bears a fairly constant relation (2·8:1) to the quantity of nitrogen present in the urine. Deviations from this numerical relation are usually found to occur shortly after the operation and also shortly before death. The two chief factors that have to be considered in explaining the former deviation are, first, the gradual increase in the severity of the diabetes, and, secondly, the condition of the animal prior to the operation. In badly nourished animals the normal relation, 2·8 parts of glucose to 1 part of nitrogen, was only gradually reached; while in well-nourished animals having presumably a considerable quantity of glycogen stored in the liver and muscles, the quantity of sugar excreted increased much more rapidly, attaining temporarily in some cases values much exceeding the normal relation of 2·8:1. The most probable explanation of the diminished excretion of sugar that occurs shortly before death is that the production of sugar from proteid is disturbed owing to the fall in the nutrition and strength of the animal. The production of sugar by the organism, in so far as it occurs at the expense of the proteids, is to be regarded as a very complex process, in which synthetic as well as hydrolytic and oxidative



processes play a part, so that one can readily understand that this function may be much interfered with by any failure of the vital powers of the organism, such as that occurring during fever or at the approach of death.

If the pancreas be only partially excised, the severity of the diabetes produced varies greatly according to the amount of gland substance left intact. If  $\frac{1}{8}$  to  $\frac{1}{12}$  of the gland substance be left untouched, an alimentary glycosuria is the only result. If the excision be somewhat more complete, a diabetes of medium severity results. The diabetes following incomplete excision tends to gradually increase in severity, probably as the result of atrophic changes occurring in the portion of gland left untouched. Exact numerical data with regard to the amount of gland necessary to prevent the occurrence of diabetes are wanting.

If a portion of the pancreas be transplanted to the subcutaneous tissue of the abdominal wall, and the remainder of the gland be then excised, it is found that no glycosuria occurs in those cases in which the transplantation has been completely successful. Occasionally the operation is followed by a slight transitory glycosuria. Diabetes of the severest type immediately follows excision of the transplanted portion of gland. Ligature of the vessels supplying the transplanted piece of gland also induces diabetes; while no permanent glycosuria follows ligature of its veins. Minkowski considers the latter fact to be important as an indication that the direct passage of blood from the transplanted portion of gland into the portal vein is not necessary for the prevention of pancreatic diabetes.

It may be concluded from these results that there is no direct relation between the external secretion of the pancreas and that function which regulates the metabolism of glucose. A consideration of these facts naturally leads to the question as to whether these two apparently independent functions are dependent upon the activity of the same cellular elements. Schäfer has suggested that the specific function of the pancreas in the metabolism of sugar is controlled by the cells in the cell islets of Langerhans. As evidence in favour of this view may be adduced the fact that diabetes has in several cases been found to be associated with degenerative changes in the epithelial cells of the islets of Langerhans without any other destructive pathological lesion of the pancreas.

*Relation of Diabetes Mellitus to lesion of the Pancreas.*—Opie has described in a paper (*Journal of Exp. Med.* vol. v. March 25) changes in the Langerhans islets in various forms of chronic interstitial pancreatitis. So long as the fibrous tissue overgrowth did not penetrate within the lobules, the islets were not affected and diabetes did not ensue. In two out of three cases of interstitial pancreatitis, diabetes mellitus was present. Opie also, in a previous paper, described a peculiar lesion of the pancreas in a case of diabetes mellitus where the islets were replaced by areas of hyaline material. He describes the reactions of this material to different stains. The description of the case in his most recent paper is very interesting, showing that diabetes mellitus may occur when the ordinary secretory cells are normal and the Langerhans cells degenerated.

In order to obtain an insight into the nature of the disturbance of metabolism, which underlies pancreatic diabetes, it is important to consider the fate of the different forms of carbohydrates within the organism of the diabetic animal. Glucose and its anhydrides, in so far as the latter undergo digestion, are absorbed and excreted as glucose without apparently undergoing any change within the organism. It is true that the increased excretion of glucose by the diabetic animal, after a diet rich in polysaccharides of glucose, does not exactly correspond to the quantity of



glucose absorbed as estimated by the difference between the amount of polysaccharides present in the food and that passing out in the fæces. In all probability this deficit is to be explained not by the supposition that a part of the glucose is utilised within the diabetic organism, but by the hypothesis that part of the carbohydrate within the intestine is decomposed by organised ferments into lactic acid, butyric acid, carbon dioxide, and other products. The absence of the pancreatic juice from the intestinal canal, by diminishing the digestion and absorption of carbohydrates, leads to their prolonged retention within the alimentary canal, and thus gives an opportunity for the action of organised ferments. When maltose is given to dogs after excision of the pancreas, glucose alone is found in the urine, and the amount in the urine corresponds almost exactly to the quantity of glucose derivable by hydrolysis of the maltose given.

The investigation of the fate of the lævorotatory hexose lævulose and its derivatives has led to very interesting results. When lævulose is given, only traces of it appear in the urine, while the quantity of glucose excreted is much increased, the relation of the quantity of glucose to that of nitrogen mounting in some cases from 2·8:1 to 11:1. Part of the lævulose is utilised within the body; while the remainder is converted into glucose and excreted as such. If large quantities of lævulose be given, traces of unchanged lævulose appear in the urine. On the other hand, if its anhydride inulin be given even in large doses, no lævulose appears in the urine, presumably on account of slow conversion of inulin into lævulose within the alimentary canal, so that large quantities of lævulose are not present there at any given time. Cane-sugar is inverted by an intestinal enzyme, and acts in the same way as the equal parts of glucose and lævulose derivable from it.

After the introduction of lactose into the alimentary canal of diabetic dogs, only glucose in increased amount is found in the urine, none of the lactose being excreted unchanged. Galactose also produces an increased excretion of glucose. None of it is excreted unchanged.

With regard to the changes in the fluids and organs of diabetic dogs, the following are the chief facts. The quantity of sugar in the blood is markedly increased, reaching values of from ·3 to ·4 per cent. If, after extirpation of the pancreas, the kidneys be also excised, the percentage of glucose in the blood is found to rise to ·6 to ·7. This result is most simply explained by a lessened utilisation of sugar within the body, in consequence of which the cessation of the secretion of urine leads to the accumulation of glucose in the blood. The quantity of glycogen in the leucocytes is much increased, being four to five times as great as normal. This increase is the more interesting in view of the absence or diminution of glycogen in the liver.

In the severest form of diabetes consequent upon complete extirpation of the pancreas, only traces of glycogen are to be found in the liver a few days after death. It is obvious that the absence of glycogen from the liver is not due to any want of material for its formation, since the quantity of glucose in the blood is largely increased, and a diet rich in starch does not perceptibly increase the quantity of glycogen in the liver. These facts favour the view that there is an intimate relation between the absence or diminution of glycogen in the liver and the accumulation of glucose in the blood. If lævulose be given after excision of the pancreas, the quantity of glycogen in the liver is distinctly increased. The glycogen formed under these conditions was found to be identical with that deposited in the liver of a normal animal yielding on hydrolysis with dilute acid the dextro-



rotatory glucose. The quantity of glycogen in the muscles is also increased by the administration of lævulose.

The muscles of dogs after extirpation of the pancreas apparently contain less lactic acid than the muscles of a normal animal. The quantity of glycogen is also diminished, but not so markedly as in the case of the liver.

The quantity of urine passed and the total nitrogen excreted are markedly increased. The presence of acetone, aceto-acetic acid, and  $\beta$  oxybutyric acids is inconstant, their occurrence in the urine after excision of the pancreas being indeed the exception rather than the rule. Usually they are met with at a late stage of the disease, when the animal shows marked disturbance of nutrition, indicated by great loss of weight and strength. The largest amount of  $\beta$  oxybutyric acid found in the urine of twenty-four hours was 4 gms. It must, however, be borne in mind that the quantity of  $\beta$  oxybutyric acid excreted probably represents only a fraction of that formed within the organism; for even after complete excision of the pancreas the body is able readily to oxidise  $\beta$  oxybutyric acid. This has been proved by the fact that after administration of  $\beta$  oxybutyric acid to a diabetic dog only  $\frac{1}{20}$  part of the quantity given could be recovered from the urine. Although the greater part of the acid appears to undergo complete oxidation, some is also excreted in the form of aceto-acetic acid and acetone.

A large number of different theories has been suggested to explain the rôle played by the pancreas in the utilisation of the glucose formed within the body, and the problem is to be regarded as still far from completely solved. The experimental evidence already adduced proves that the reason for the diabetes following excision of the pancreas is not the absence of the pancreatic juice from the intestines; but that the diabetes must be referred to a disturbance of metabolism produced by the withdrawal of some influence exerted by the functionally active gland. Two views with regard to this influence naturally suggest themselves. The pancreas either renders innocuous some substance whose retention within the body produces the accumulation of sugar in the blood, or it is normally directly or indirectly concerned in promoting the utilisation of glucose within the body.

In order to test the former hypothesis, Minkowski injected the blood of a diabetic dog into a healthy one. From the negative result obtained no conclusion could be drawn; for the healthy dog, possessing a functionally active pancreas, was presumably in a position to render innocuous the hypothetical toxic substance present in the blood of the diabetic dog. Hédon attempted the solution of the same problem by a slightly different method. He injected the blood of a dog suffering from the severest type of diabetes into one from whom the pancreas had been partially removed, and whose urine, when the animal was maintained on a meat diet, contained only .67 per cent glucose. After the injection, no increase in the quantity of glucose excreted by the dog whose pancreas had been partially excised was observable. The latter experiment, although not absolutely decisive, still renders improbable the conclusion that the diabetes following excision of the pancreas is due to the retention of a toxic substance.

Under the head of the second theory, that the diabetes occurring after excision of the pancreas is due to the withdrawal of some influence normally exerted by the pancreas on the metabolism of carbohydrates within the body, several hypotheses have been brought forward. The pancreas may prevent the accumulation of glucose in the blood by either favouring its katabolism in the tissues or by promoting its conversion into glycogen by



the liver. It may favour oxidation either by direct action on the glucose, rendering it more accessible to oxidation by the tissues, or by secreting some substance to be carried by the blood-stream to the tissues and utilised by them in the oxidation of glucose. Sufficient evidence is not available to test either of these possibilities. Some observers have recently endeavoured to prove that pancreatic diabetes is due to the absence of a glycolytic ferment. Claude Bernard discovered that after death the quantity of sugar in the blood diminished more or less rapidly. Later observers showed that this diminution took place even when micro-organisms were excluded, and ascribed the action to a glycolytic enzyme whose activity was destroyed at 54° C. This enzyme has been shown to act as an oxidising agent, and is held by some observers to be a nucleoproteid. Lépine states that it is primarily derived from the pancreas, passes thence into the blood, and is carried by the leucocytes. Lépine states that it is present in the blood only in small amount after extirpation of the pancreas, and ascribed the resulting diabetes to the diminution of glycolytic ferment in the blood. Several objections to this view have been raised. The most important of these is that of Minkowski, who showed that the quantity of the glycolytic enzyme is the same in the blood of diabetic as in that of normal dogs.

The theory previously referred to—namely, that the pancreas acts by aiding the liver and other organs in the building up of glycogen from glucose—has more evidence in its favour. It has already been shown that the conversion of glucose into glycogen does not take place in a dog from which the pancreas has been removed. If this conversion be necessary for the further utilisation of glucose within the body, it is easy to understand that glucose must accumulate in the blood when the pancreas has been removed. For the formation of glycogen from lævulose the influence of the pancreas is unnecessary. This difference in the behaviour of glucose and lævulose is still quite inexplicable.

Lastly, the question as to whether the function of the pancreas in carbohydrate metabolism is a specific one may be shortly considered. Some observers have stated that excision of the salivary glands of the dog also produces diabetes. The glycosuria so produced is, however, slight, transitory, and not constant, and is probably to be classed with the transitory glycosuria sometimes occurring after operations on the human subject. If the pancreas be excised along with the salivary glands, the diabetes that results is not more severe than that following excision of the pancreas alone. Similar statements hold good with regard to the glycosuria occasionally following excision of the thyroid (see also "Diabetes," vol. ii., and "Glycosuria," vol. iv.)

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### Pancreas, Diseases of.

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THOUGH diseases of the pancreas have been looked on as uncommon, it is only because of the position of the organ deeply at the back of the abdomen, and, therefore, of the difficulty in making a physical examination of it during life, unless considerably enlarged, that it has not received more attention; and this clinical want of attention has, unfortunately, until quite recently, generally been carried to the post-mortem room, otherwise we should have had more notice taken of the subject. Dr. Hale White has given a record of all the cases of pancreatic disease occurring in the post-mortem room of Guy's Hospital during fourteen years, from 1884 to 1897, showing that out of 6708 autopsies the pancreas was found diseased or injured in 142; that is, out of all cases dying in a large general hospital, 2 per cent had disease of the pancreas. This goes to prove the view expressed at the International Medical Congress held in Paris, that pancreatic diseases are far from being uncommon; and, fortunately, this recognition of the importance of the subject is likely to have more than a mere pathological interest; for although surgery can hold out little probability of being able to materially benefit malignant disease, much can be done for the cure of both cystic and inflammatory affections.

ANATOMICAL CONSIDERATIONS.—The pancreas is a compound racemose gland, retroperitoneal in position, it being placed in front of the first lumbar vertebra with the head embraced by the concavity of the duodenal curve, and the tail reaching as far as the hilum of the spleen. It is of irregular shape, consisting of a body, head, and tail, the junction between the head and body being somewhat constricted, so as to form a neck. Between the head of the pancreas and the duodenum are the superior and inferior pancreatico-duodenal arteries. The superior mesenteric artery and vein emerge from beneath the body of the pancreas and then pass over the transverse portion of the duodenum. There is sometimes a prolongation of the pancreas upwards from the head; this is called the lesser pancreas. The pancreas weighs about 3 ounces, and on section is of a pinkish-cream colour. It has in front of it the meso-colon and stomach, and it lies upon the aorta, vena cava, left crus of the diaphragm, and left suprarenal capsule. Its own capsule is of delicate areolar tissue, and sends processes between the lobules of the gland. The pancreatic duct (D. of Wirsung) begins near the tail and passes towards the right rather nearer the lower and posterior than the upper and anterior borders of the gland. It receives in its course branches from the different lobules which enter it at right angles. It is a thin white tube, which at its termination curves downwards in relation with the common bile-duct to enter the descending portion of the duodenum into which it opens. When there is a lesser pancreas its duct joins the proper pancreatic duct before its termination. A small accessory pancreatic duct (D. of Santorini) is occasionally present; and collecting the secretion from the upper portion of the head of the gland, it opens separately and directly into the duodenum about an inch and a half above the entrance of the ordinary duct. The pancreas receives its blood-supply from the superior and inferior pancreatico-duodenal arteries on the right, and the pancreatica magna and pancreaticæ parvæ branches of the splenic artery on the left. The veins empty into the splenic and superior mesenteric. The lymphatic vessels pass to the lumbar lymphatic glands. The nerves are derived from the solar plexus.

PHYSIOLOGICAL CONSIDERATIONS.—The pancreatic fluid serves to emulsify and split up fat into the fatty acids and glycerine, to convert starchy matters into dextrine and grape sugar, and albuminous and gelatinous substances into peptones; but besides secreting pancreatic fluid, the pancreas



also throws off something into the blood necessary for the transformation of carbohydrates, and in the absence of this pancreatic extract from atrophy or other destructive disease of the pancreas; diabetes results.

**PATHOLOGICAL CONSIDERATIONS.**—When it is borne in mind that the pancreas is a racemose gland with its various channels opening into a duct, which opens along with the common bile-duct into the second part of the duodenum, a channel usually containing septic organisms, and frequently liable to catarrh and other disorders, it is not surprising that inflammation should frequently occur in it. Just as in the liver we may have acute and chronic catarrh of the ducts, infective and suppurative cholangitis, and inflammation of the interlobular tissue ending in cirrhosis, so in inflammation of the pancreas we may have any one or more of those affections, though as yet we have not learnt to recognise acute or chronic catarrh of the pancreatic ducts apart from jaundice, or infective and suppurative inflammation of the ducts apart from abscess of the pancreas itself. As our means of diagnosis become more perfect, diseases of the pancreas will be more frequently recognised and awarded their proper place in medicine.

I have seen cases of discomfort with some swelling at the epigastrium associated with dyspepsia and ague-like attacks, but without jaundice, or with only very slight jaundice, which I thought might be explained on the hypothesis of infective inflammation of the pancreatic duct, the cases having cleared up under general treatment. I have also seen the same symptoms associated with more pain, irregular fever, more marked swelling and tenderness over the pancreas, and discharge of pus by the bowel from time to time, but without any collection sufficiently large to form a distinct abscess.

In the case of obstructive disease in the bile-ducts we have ocular evidence in the shape of jaundice, but in pancreatic disease the characteristic sign of fat necrosis only becomes manifest when the abdomen is opened; glycosuria, fat in the stools, and lipuria occur too seldom to be of much use, but when present they are of great diagnostic import.

The essential and immediate cause of the various forms of pancreatitis is bacterial infection, this having been positively proved both clinically in the human subject and experimentally in the lower animals; but as in inflammatory affections of the liver and bile-ducts we look for extrinsic causes, so in pancreatic diseases we find biliary and pancreatic lithiasis, injury, gastro-duodenal catarrh, ulcer, and cancer of the stomach, pylorus, or duodenum, and zymotic diseases, such as typhoid fever and influenza, to be determining factors; though in some cases pancreatitis has come on suddenly in persons in robust health, and the determining cause has been beyond recognition. Though the infection may arise from the blood as in pyæmia, or by direct extension from the neighbouring tissues, as in ulcer of the stomach, yet the usual channel is through the duct, as in the cases arising from gall-stones in the common duct, and from gastro-duodenal catarrh, that I related in my lecture at the London Polyclinic in June 1900.

*The Relation between Gall-stones and Pancreatitis.*—The association of the two conditions has been mentioned by several observers; the connection between the two was absolutely forced on my mind by the frequency with which I found inflammatory enlargement of the head of the pancreas when operating for gall-stones in the common duct, my views on this subject, supported by a number of cases observed during several years since 1892, having been brought forward at the International Congress held in Paris in 1900.

In the *British Medical Journal*, No. 14, 1896, Dr. Kennan described a



case of acute pancreatitis ending fatally on the second day in a woman of 38, and at the post-mortem a large number of gall-stones were found in the common duct, one being partially extruded into the duodenum.

Körte (*Deutsch. Chir. Stuttgart*, 1898) notes that lesions of the pancreas are frequently associated with disease of the bile-ducts.

Lancereaux (*Traite des maladies des foie et des pancreas*, 1898) mentions the possibility that a gall-stone impacted in the diverticulum of Vater may occlude the pancreatic duct, and produce conditions favouring the entrance of organisms into that duct.

Dr. Opie, in the *American Journal of Medical Science* for Jan. 1901, p. 27, relates cases, one of Dr. Osler's, and several from various sources, showing the relation between pancreatic and gall-stone troubles.

In several of my cases gall-stones were not found, but only old and firm adhesions with an antecedent history of paroxysmal attacks followed by jaundice, which afforded strong presumptive evidence that cholelithiasis had been present at some time; and it seems quite possible that a temporary occlusion of the common duct with the damming back of infective secretion or with injury to the ducts may set up a pancreatitis, which may itself then cause compression of the common bile-duct, and so lead to a perpetration of the trouble started in the first instance by an impacted gall-stone.

*Fat necrosis* is commonly found in association with pancreatitis and other diseases of the pancreas, and the relation between the two conditions has given rise to much speculation; but the facts that fat necroses are not found in all acute pancreatic diseases, and that they have been frequently noted during abdominal operations for other diseases, and in autopsies where there was no suspicion of pancreatitis, appear to show that the condition giving rise to it is not essentially a pancreatitis as suggested by certain authors, though these facts do not disprove that fat necrosis is essentially due to interference with the discharge of pancreatic secretion, and so to an escape of pancreatic fluid into the tissues, whence it may be taken up by the lymphatics or blood-vessels. Flexner (*Contribution to the Science of Medicine*, Johns Hopkins press, Baltimore, 1900) regards the fat necrosis as the effect of the fat-splitting ferment of the pancreatic fluid which has in some way escaped from the duct into the surrounding tissues. It is difficult at first sight to explain the patches of fat necrosis occurring at a distance from the pancreas, for instance, in the pericardium, unless it be by absorption of the ferment, and its diffusion by means of the lymphatics, though it is easy to understand and undoubtedly does explain the disintegration of the fatty tissues immediately surrounding the pancreas, and extending by continuity first to adjoining, and then even to distant, parts in the abdomen and thorax. By fat necrosis is understood a splitting up of the fat into fatty acids and glycerine; the latter is absorbed, but the former, being insoluble, remain in the cells and unite with calcium salts, forming white patches of various sizes in the subperitoneal fat and in the omentum and mesentery. It was first described by Balser in 1882, but has been since investigated by Langerhans, Hildebrand and Dettner, Milisch, Williams, and Opie. Experiments by Opie, who ligatured the pancreatic ducts in the cat, go to show that widespread fat necrosis may be expected to follow very rapidly.

*Hæmorrhage in Pancreatic Diseases.*—It is well known that hæmorrhages into the pancreas may occur apart from injury, and that, although they may be recovered from, as shown by the remains of extravasated blood in the gland in persons dying from other diseases, yet such spontaneous



hæmorrhages may lead to death from collapse either immediately or after some hours.

Curiously, this may occur in persons apparently in good health, and without any premonitory signs on which a diagnosis can be based, the only symptoms at the time being those of collapse with dyspnœa and feeble pulse.

It is also well recognised that a hæmorrhagic condition co-exists with cancer of the head of the pancreas. Some years ago I thought this to be altogether dependent on the cholemia, until increased experience in operations on deeply jaundiced subjects has taught me that there is much less danger of serious hæmorrhage in patients jaundiced from gall-stones than in those where the jaundice depends on pancreatic disease. In several cases of cancer of the pancreas on which I have operated, the bleeding has been the immediate cause of death; in one aged subject especially was this well marked after a cholecystotomy for the relief of jaundice. Death occurred on the third day from collapse, and at the autopsy there was found extensive effusion of blood away from the site of operation and behind the peritoneum, extending into the loins, around the kidneys, and into the cellular tissue beneath the diaphragm, the blood being in sufficient quantity to account for death; and though the bleeding had evidently arisen from the pancreas, there was no manifest vascular lesion to account for the hæmorrhage. In another case, sent to me in 1888 by Professor Clifford Allbutt, a cholecystotomy was followed by persistent oozing of blood from the interior of the gall-bladder and from the stitch punctures, which resisted all then known remedial measures in the way of styptics, pressure, transfusion, etc., and proved fatal on the ninth day. In neither of these cases was there any peritonitis or other cause than the hæmorrhage to account for death. Before operating on these cases I now always administer chloride of calcium in 20 to 30 grain doses every four hours from twenty-four to forty-eight hours previous to operation, and by enema in 60-grain doses for forty-eight hours afterwards.

These arguments are brought forward to show that there is some as yet ill-understood relation between pancreatic disease and serious hæmorrhage; but I cannot help thinking that it is a mistake to allow this fact to influence our views on the pathology of inflammation by adopting permanently the name hæmorrhagic pancreatitis in cases where there has been no bleeding, or no more hæmorrhage than occurs frequently in inflammation of other parenchymatous organs, though it may perhaps be useful to retain the name for those cases of pancreatitis associated with well-marked interstitial hæmorrhage, and in which the disruption of the gland may actually be the immediate cause of the inflammation.

I have recently read a very interesting paper in the *Boston City Hosp. Report* for December 1900, by Dr. F. B. Lund, entitled "Acute Hæmorrhagic Pancreatitis and its Surgical Treatment: with a Report of Six Cases," yet in remarking on the second case, the author says, "This case is notable for the absence of pancreatic hæmorrhage." In the fifth case there was the evacuation and drainage of a small abscess of the pancreas, and no evidence of hæmorrhage, and in the sixth case also there is no evidence whatever of hæmorrhage. The title of the paper is surely a misnomer, yet other authors adopt the same nomenclature in the same indefinite manner.

Dr. Flexner produced an inflammation of the pancreas associated with hæmorrhage within forty-eight hours by injecting the bacillus pyocyaneus and the bacillus diphtheriæ into the pancreatic duct in animals (*Contributions to the Science of Medicine*, Baltimore, 1900, p. 743). Hildebrand



(*Centralblatt für Chirurgie*, 1895, Band xxii. p. 297) suggested that the hæmorrhage in acute pancreatitis was due to trypsin. Hlava produced hæmorrhagic pancreatitis by injecting the bacillus diphtheriæ into the pancreas.

The only conclusions we can come to, after carefully considering the whole subject, are:—

That in certain diseases of the pancreas there is a general hæmorrhagic tendency which is much intensified by the presence of jaundice, but that hæmorrhage may occur into the pancreas unassociated with inflammation, or with jaundice, or with a general hæmorrhagic tendency.

That both acute and chronic pancreatitis can and do frequently occur without hæmorrhage, but that some cases of pancreatitis are associated with hæmorrhage.

That inflammation of the pancreas may be more conveniently and scientifically classified like inflammation of other organs, as acute, subacute, and chronic, and there is no reason to use the term hæmorrhagic pancreatitis except as a variety of acute pancreatitis, the hæmorrhage being merely an accident in the course of the disease.

### PANCREATITIS

*Symptoms.*—It seems to me convenient to consider pancreatitis clinically under the headings of (1) Acute, often associated with hæmorrhage and gangrene; (2) Subacute, often associated with suppuration; and (3) Chronic, associated with an increase of the interstitial tissues. The symptoms are very variable, and differ in the separate forms. It is a disease without pathognomonic signs, and a correct diagnosis is usually only arrived at by a careful study of the history, mode of onset, and combination of symptoms.

*Acute Pancreatitis.*—This is usually ushered in by a sudden pain in the superior abdominal region, accompanied with faintness or collapse, and followed sooner or later by vomiting. It is almost constantly accompanied by constipation, so that it is quite usual for these cases to be mistaken for intestinal obstruction at first. The obstruction, however, is not absolute; flatus passes, and a large enema may secure an evacuation; if the patient survive for several days, diarrhœa may supervene. The pain may be so severe as to produce syncope or collapse; and though the pain does not quite pass away, it has a tendency to be paroxysmal, and to be increased by movement; it is associated with well-marked tenderness just above the umbilicus, or between it and the ensiform cartilage. The pain is soon followed by distension in the superior abdominal region, which may become general, and usually does in the later stages, and by vomiting, first of food, then of bile. The vomiting may be severe, and each seizure may aggravate the pain, but at times vomiting may not be a prominent symptom. Slight icterus from associated catarrh of the bile-ducts is usually present, and deepens the longer the patient survives. The aspect is anxious and the face is pinched, resembling the facies of peritonitis, which in fact may be present. The pulse, which is rapid and small, is a better guide than the temperature, which may be normal, subnormal, irregular, or high. Delirium comes on in the later stages. The distension, pain, and tenderness prevent an exact examination of the pancreas, which would otherwise be found enlarged. Death usually supervenes from the second to the fifth day from collapse, though in the less acute cases life may be prolonged for days or weeks. Acute infective pancreatitis thus takes on the form of acute peritonitis starting in the superior abdominal region. If life be prolonged, the case



comes under the category of subacute pancreatitis, and in that case the onset is usually less grave, though often equally sudden.

*Post-mortem Appearances.*—Post-mortem appearances are variable, but the following are fairly characteristic:—As a rule areas of fat necrosis will be observed in the omentum and in the fatty tissues in the neighbourhood of the pancreas, but they may be found all over the abdomen. The peritoneal cavity may contain blood-stained fluid. In the situation of the pancreas in the hæmorrhagic form will be found a large, hard, dark, chocolate-coloured mass several times the size of the normal pancreas. On section of this very little trace of normal pancreatic tissue may be discovered, the whole organ being infiltrated and distended with fluid. Dark brown or black necrotic areas can easily be seen, and sometimes in the gangrenous form the whole of the pancreas is converted into a black slough lying in the midst of dirty, gray, thin fluid, with very offensive odour. The adjoining tissues and organs will in these cases be found stained and dark-coloured. It is very common to find gall-stones in the common duct or in the gall-bladder.

*Treatment.*—In acute infective pancreatitis treatment practically resolves itself into that of peritonitis commencing in the superior abdominal region. The pain at the outset is so acute as to necessitate the administration of morphia, and the collapse will probably demand stimulants, which on account of the associated vomiting may have to be given by enema. In the early stages the symptoms are so indefinite that the indications for surgical treatment are often not clear enough to warrant operation, and until the collapse has passed off no surgical procedure would be justifiable. The simulation of intestinal obstruction will probably lead to efforts to secure an evacuation of the bowels and relief to the distension. Just as in perforative or gangrenous appendicitis an early evacuation of the septic matter is necessary to recovery, so in this equally lethal affection an early exploration from the front through the middle-line above the umbilicus, or from behind through the left costo-vertebral angle, is demanded in order to evacuate the septic material and adopt free drainage. The after treatment will be chiefly directed to combating shock and keeping up the strength until the *materies morbi*, both local and general, can be thrown off.

*Subacute Pancreatitis.*—This may have a sudden onset, with acute pain and vomiting associated with constipation, but the collapse is not so marked and may even be absent; the upper abdominal region does not become so rapidly swollen, and vomiting is less severe and less prolonged. At other times the onset is more gradual, though the symptoms may be similar. Tenderness over the pancreas is well marked, and on account of the tympanites being less than in the acute form it may be possible to feel the swollen gland, especially under an anæsthetic. Constipation gives place to diarrhœa, and pus or blood may be noticed in the stools, which have a very foetid odour. The pulse will be less rapid and less thready than in the acute form, and the temperature is more irregular. I have seen the temperature reaching 104° and 105° F., and yet the pulse to vary between 90 and 110. The morning temperature may be normal, and the evening temperature high for several days or even weeks. Rigors may occur and may be repeated from time to time. The pain occurs in paroxysms, but there is also a constant dull pain at the epigastrium. The patient may lose the more urgent symptoms and appear to be really improving, but the loss of flesh and feebleness continue, and relapses usually occur, leaving the patient each time more and more feeble, until death supervenes from asthenia. Albuminuria is pretty constant, but glycosuria is rarely present, and lipuria is an uncommon symptom. As the disease is less intense than in the fulminating form, suppuration usually occurs.

If an abscess develop, the pus may form a tumour projecting in the



superior abdominal region, forming a tender swelling behind the stomach, or perhaps coming to the surface above or below that viscus; or it may burrow into either loin, forming a perirenal abscess, or passing under the diaphragm it may form a subphrenic abscess.

Occasionally the pus may follow the psoas muscle and form a subperitoneal abscess in the iliac region, or even passing over the brim of the pelvis it may collect in the left broad ligament. I have seen these several terminations in cases either under my own care or under the care of colleagues, and I have operated four times for abscess of the pancreas. Sometimes the abscess bursts into the stomach and is vomited, or into the bowel and is voided per anum, after which diarrhœa may continue, and pus may be seen from time to time as any fresh collection forms and bursts. I have seen both of these methods of evacuation of the pus. With the evacuation of the abscess relief occurs for a time, and the temperature improves; but relapses usually occur, and a mild form of septicæmia persists, with a hectic temperature. Death is the usual termination, but recovery may occur after a tedious and prolonged illness, as in the case now related.

On 17th March 1900 I was asked by Dr. J. Glen of Middlesborough to see a female patient, aged twenty-six years, when I obtained the following history:—The patient was first attended the last week of December 1899. Her symptoms then indicated a chronic form of dyspepsia, with recurrent attacks of diarrhœa, the latter associated with indurated fæces. No relief from treatment resulted. The main symptoms at this time were general tympanitic condition of abdomen, with tenderness over the sigmoid flexure, no acceleration of pulse, and no rise of temperature. About the second week in January 1900, the diarrhœa still persisting, a good deal of fresh blood was present with each evacuation, thus giving one the idea of dysentery. Then retching and vomiting set in, but this condition was probably set up by the free exhibition of powder of ipecacuanha. This state continued more or less for upwards of a fortnight, when improvement set in. The tympanites disappeared, the tenderness of the left groin became less, there was less sickness, and desire for food returned. The diarrhœa abated, there being only one or two evacuations in the twenty-four hours, the motions being formed. The improvement continued till the second week in February. The patient at this time, having gained flesh, was daily gaining strength, and was able to sit up in her dressing-gown for a time each day. A relapse took place, accompanied with much pain in the epigastric region, constant sickness, return of diarrhœa with blood in the stools, rapid emaciation, and quick pulse with rise of temperature. This condition persisted in spite of all treatment. There had never been blood in the vomited matter except once, when there was a slight streak, as if it might have come from the pharynx during the act of severe retching. Since the relapse there had developed a dull area with tenderness over the pancreas, but the degree of tenderness varied from day to day. At one time there was retention of urine, afterwards incontinence; no albumin or sugar was present. Hæmorrhage was very profuse, the blood being gruel-like and mixed with stools which were very offensive. Dr. Glen showed me the report of the Clinical Research Association of an examination of the fæces, which contained some blood and pus, but no cells suggestive of growth, and no tubercle bacilli. When I saw the patient she was looking very ill and emaciated, with feeble, quick pulse; there was tenderness in the superior abdominal region, with rigid recti, but none along the course of the colon either on the right or left side. Examination under an anæsthetic revealed a swelling in the situation of the pancreas, but no fluctuation could be made out. As there was resonance



in front of it, it was clearly behind the stomach, and from its position it was above the colon. Before the anæsthetic the patient said there was very great tenderness on pressure over the swelling in the epigastrium. The rectal examination was negative, except that there was ballooning. We made a diagnosis of suppurative pancreatitis, the abscess having discharged into the bowel. I advised five grains of salol, two grains of quinine, and half a grain of opium, to be given thrice daily, the patient to be watched with a view to the testing of her temperature, pulse, etc. Plasmon and somatose dissolved in broth, tea, etc., were ordered, as the patient could not take milk. The question of operation was raised and dismissed, to be further considered in about a week or ten days, as she was at the time too feeble to bear even an exploratory incision. From this time improvement steadily occurred, and when I saw her again in a fortnight she was picking up distinctly, though there was still well-marked tenderness over the pancreas. In June she had gained nearly a stone and a half in weight, looked much better, and could take her food well; there was, however, still a little rigidity of the recti and some tenderness, though no distinct tumour of the pancreas. The diarrhœa had disappeared, and there was neither pus nor blood in the motions.

*Post-mortem Appearances.*—In suppurative pancreatitis, which is for the most part subacute, there may be a diffuse infiltration of pus throughout the gland, the ducts being full of muco-pus, and suppuration being found in the gland along the course of the excretory channels, penetrating to the inter-cellular space, and forming separate collections of pus in a similar way to that which takes place in the liver in suppurative cholangitis. If the pancreatitis is limited to one or more regions of the gland the pus may then collect and form an abscess of variable size composed of broken-down tissue with disintegrated fat and pus, and it is not unusual in these cases to find hæmorrhage into the abscess cavity giving the pus a chocolate colour, though at times there may be an absence of blood, and the pus may then be laudable.

There is usually peripancreatitis, and it is common to find effusion of a thin grayish fluid containing pus cells in the lesser peritoneal cavity. A localised abscess may project into the lesser cavity of the peritoneum, and passing forward it may tend to point above the stomach. In other cases a leakage of pus may occur into the peritoneal cavity, producing peritonitis, at first of the lesser and later of the greater peritoneal sac. If the abscess bursts behind the peritoneum it may burrow backwards and form a perirenal abscess; or passing further in front of the psoas sheath it may either simulate an iliac abscess pointing above Poupart's ligament, or proceeding farther towards the pelvis it may form a pelvic abscess. I have seen and operated on all these forms. In three cases I have known the pus to burst into the stomach and into the bowel.

*Treatment.*—The subacute form of pancreatitis is much more amenable to treatment, as the indications are so much more definite, and there is more time for careful consideration; and though it has usually only been attacked when an abscess has formed, and is manifestly making its way to the surface, yet there is no reason why in some cases surgical treatment should not be adopted at an earlier stage. As in the acute condition, morphia may be required to relieve the paroxysmal pain, and stimulants and food by the rectum to relieve the collapse and support the strength. The distension, if present, may also demand attention, and may have to be relieved by lavage of the stomach and turpentine enemata, or by the administration of calomel by the mouth. Calomel is also of benefit by acting as an intestinal antiseptic, for which purpose it may be given in small repeated doses, or in doses of five grains, followed by a saline aperient. As soon as the constipation is relieved diarrhœa is apt to supervene, when salol and bismuth, with small doses of opium, may be given. If surgical treatment is decided on, a median incision above the umbilicus will enable the operator to palpate the



pancreas and locate any incipient collection of pus, which, if practicable, should be evacuated by a posterior incision in the left or right costo-vertebral angle; or failing that possibility, the collection of pus may be aspirated and the cavity opened and packed with gauze, which may be brought forward through a large rubber drainage-tube, which will in the course of from twenty-four to forty-eight hours establish a track isolated from the general peritoneal cavity. In one case I was able to do this, but the operation was undertaken at too late a stage to be successful; and though the patient lived two or three days afterwards, the evacuation of the pus seemed to make very little difference to the general septic condition previously existing, and death occurred from increasing debility on the fourth day. The method adopted had, however, been successful from the point of view of drainage, and the track of the gauze and tube was isolated from the general peritoneal cavity. If a definite abscess form and approach the surface in front or in either loin, the treatment will be that of incision and drainage, as in the case of any other abdominal abscess. Of four cases on which I have operated two recovered completely, one recovered from the operation, but died a few weeks later from debility. The fourth case is the one just referred to. The strength must be maintained by careful feeding and the judicious administration of stimulants. It will be necessary to keep a sharp look-out for further collections of pus and for subphrenic abscess or empyema, which on recognition will need treatment.

*Chronic Pancreatitis.*—Chronic pancreatitis, at first hypertrophic, but later cirrhotic, is usually considered to be a very rare disease, but experience would lead me to believe it to be a much more common affection than the acute or subacute forms, and I have no doubt that some cases put down as malignant disease of the head of the pancreas and terminating fatally may be cases of chronic interstitial pancreatitis. My reason for this belief is shown in the cases related below, selected out of twenty cases on which I have operated. My experience has resulted from my having operated on a large number of cases of jaundice depending on obstruction to the common bile-duct; the obstructive jaundice, wasting, paroxysmal attacks of pain, and ague-like seizures having given rise to the suspicion of gall-stones, and the absence of relief by medical treatment having rendered surgical treatment necessary. My first case of this nature, which occurred in 1892, was a revelation to me, as the patient was extremely ill before the operation, which was in fact undertaken too late; and as there was the opportunity of a post-mortem examination, the absence of malignant disease or of other cause than the chronic pancreatitis and associated jaundice to account for the death was directly proved.

This form of pancreatitis is quite distinct from the acute or subacute form, though I think it is probable that some of the latter cases end in chronic interstitial pancreatitis. It usually arises by extension of the inflammation from the pancreatic ducts to the interlobular tissues, and is generally dependent on gall-stones in the common duct, which lead to the entrance of organisms from the duodenum that multiply in the retained secretion both in the pancreatic and bile ducts, and so set up an infective cholangitis and pancreatitis. A merely temporary block in the common duct may produce pancreatitis, and the infection once started may lead to swelling of the head of the pancreas, and thus to pressure on and partial occlusion of the common bile-duct and Wirsung's duct, in this way accounting for the persistence of the symptoms after the obstruction has passed away and for the relief by drainage, as in the cases referred to in my lecture at the London Polyclinic.



Pancreatic lithiasis, or gastric, pyloric, or duodenal ulcer, may also produce interstitial pancreatitis. Its course varies. The onset may be quite gradual and painless, or may be ushered in by a severe pain at the epigastrium, followed by jaundice resembling a gall-stone attack, and associated with nausea and vomiting, and perhaps followed by a feeling of chilliness, or even a rigor. The pain, however, is not over the gall-bladder, and does not pass round the right side to the subscapular region, but is central, and passes backwards to the midscapular region or round the left side, thus resembling stomach rather than gall-bladder pain. The tender spot is usually an inch above the umbilicus in the middle line, and not over the gall-bladder, as in cholelithiasis pure and simple. When once jaundice has come on, it tends to deepen with each attack, until it becomes continuous and chronic. The paroxysms of pain may be repeated, more or less frequently; or there may be no paroxysmal pain, merely a dull ache, deeply seated, burning and boring in character. A swelling of the pancreas may sometimes be made out; but as the recti are rigid because of the pain and tenderness in the epigastrium, it can usually only be discovered under anæsthesia.

Loss of flesh and strength are well-marked symptoms in all cases. Vomiting may in some cases be absent, but there are usually a want of appetite and flatulent dyspepsia, and always a sense of fulness and weight at the epigastrium for some time after food. Jaundice is not necessarily present at first, though it is usually present at some stage of the disease, and is often well marked; but, as was pointed out some years ago by Dr. Walker of Peterborough, the stools are white even when the pancreatic fluid alone is absent from the intestine. Diarrhœa is often present, and the stools are offensive and may be fatty. Albuminuria is common, and glycosuria may occur, but the latter is probably only present in cases where the whole gland is affected. Fever may be absent, but in some cases the temperature runs a hectic course, always rising in the evening and falling in the morning. This is especially the case where ague-like paroxysms occur. These varieties probably depend on the character of the infection in the pancreatic and bile-ducts, and also on the amount of obstruction present. Where jaundice is present the pulse may be abnormally slow; and even when the temperature is raised the pulse-rate is not much elevated, though the character of the pulse may be poor. In the later stages, especially if the disease be associated with jaundice, hæmorrhages from the nose and the bowel, vomiting of blood, and petechiæ in the skin show marked blood degeneration, and death ensues from increasing weakness.

In the more chronic cases, especially when there is contraction of the head of the pancreas, there will be found a tumour formed by the distended gall-bladder, just as there is in cancer of the head of the pancreas, for which disease chronic interstitial pancreatitis is then apt to be mistaken and a hopeless prognosis given. In such cases the gall-bladder will be found to be distended with mucus, the bile which first filled it having become gradually absorbed, the backward pressure having prevented fresh bile from entering the ducts. This may occur so gradually as to be painless, and then the gall-bladder is free from tenderness, which is seldom the case when the distension is due to gall-stones.

*Diagnosis.*—The diagnosis of chronic interstitial pancreatitis has to be made from gall-stones in the common duct, cancer of the head of the pancreas, cancer of the liver and bile-ducts, and chronic catarrh of the bile-ducts. From gall-stones the diagnosis is of no great importance, since



the two diseases are often associated and the treatment is the same ; nevertheless, it may be possible to make a diagnosis of gall-stones by the sequence of long antecedent history of spasms without jaundice, then by a severe attack of pain followed by jaundice, and after a time by recurring pains with increase of the icterus associated with ague-like attacks. The absence of tumour is more common in gall-stones than in chronic pancreatitis, though in the latter the gall-bladder may be found contracted at times. Although in pancreatitis there is usually less pain, at times the paroxysmal attacks may be equally as severe as in gall-stone seizures. The tenderness in the latter case, however, will be over the gall-bladder, and in the former at the middle line where the swollen gland can sometimes be felt, especially if the examination is made under anæsthesia ; moreover, the radiating pain in gall-stones is towards the right, and in pancreatitis towards the left or to the mid-scapular region. In cancer of the head of the pancreas the onset is usually gradual and painless, and the disease generally occurs later in life, usually after forty years of age. It is preceded by general failure of health, and when the jaundice supervenes it becomes absolute and unvarying, and is nearly always associated with a tumour of the gall-bladder, which generally attains a large size and shows no tenderness on manipulation. The liver enlarges from the biliary stasis, but there are no nodules to be felt. In some cases of cancer of the head of the pancreas a hard nodular tumour may be found on the inner side of the distended gall-bladder. Extremely rapid loss of weight and strength with increasing anæmia, but without ague-like seizures, and very characteristic, and it is common for there to be an absence of fever with a slow, feeble pulse, and later ascites with œdema of the lower limbs. Cancer of the head of the pancreas may, however, be associated with infective cholangitis, when there will be ague-like attacks and continued fever. Cancer of the common bile-duct is rare, but when present may resemble, and is usually associated with gall-stones. In cancer of the liver the irregular enlargement, the nodular feel, the rapid deterioration of health, the less intense jaundice, and the absence of fever and paroxysmal pains will usually enable a diagnosis to be made. Chronic catarrh of the bile-ducts is usually associated with an absence of symptoms, except jaundice and some loss of flesh. The entire absence of pain and fever with the relief by treatment usually give rise to little difficulty in establishing a diagnosis. In many cases a diagnosis will only be made by an exploratory operation, then the enlargement of the pancreas can be readily felt, or if the tumour be small the head of the pancreas may be found to be hard and perhaps nodular like scirrhus cancer. If ascites be present the tumour will probably be malignant. The lymphatic glands in the lesser omentum may be enlarged in both cancer and chronic pancreatitis, but in the latter they will be discrete and soft, in the former hard and perhaps infiltrating. Adhesions are usually associated with chronic pancreatitis, but are generally absent in cancer.

CASE I.—*Chronic Pancreatitis ; Cholecystotomy ; Cure.*—A man, aged 45 years, was sent to me on 25th Oct. 1898, by Dr. J. B. Berry, of Keighley. The patient was very deeply jaundiced, and told me that he had lost a stone in weight since the onset of his illness five weeks before. He gave the history of having had attacks of pain referred to the region of the gall-bladder nine years previously, but they were unaccompanied by jaundice, and passed off after prolonged treatment. From that time onward he had been free from attacks of pain up to the onset of the present illness five weeks before, when he was suddenly seized with severe pain at the pit of the stomach and became jaundiced. The pain has recurred daily, and had been so severe as to necessitate his taking morphia.



Dr. Berry noticed a swelling in the region of the gall-bladder a fortnight after the onset of his illness, and there was all along well-marked tenderness at the epigastrium, with gradually increasing enlargement of the liver. The patient's general health rapidly failed, and the loss of flesh was well marked. When I saw him he looked pinched and ill, he was very deeply jaundiced, and the urine was loaded with lithates, but contained neither albumin nor sugar. There were well-marked tenderness at the epigastrium and a smooth tumour, which was not very tender, in the gall-bladder region; the liver was enlarged, and the edge was smooth, and could easily be felt an inch below the costal margin. A diagnosis of gall-stones in the common bile-duct was made, and the patient was admitted into the Leeds general Infirmary. The operation was performed on 27th Sept., 1898. On opening the abdomen numerous adhesions between the gall-bladder and liver and the pylorus, colon, omentum, and duodenum were found. The gall-bladder was slightly distended, but no gall-stones were felt either in it or in the cystic or common duct. There was, however, a hard nodular swelling of the head of the pancreas which at the time was thought to be malignant. In order to give relief the adhesions were detached, and the gall-bladder was drained by cholecystotomy. On Oct. 28th, I wrote to Dr. Berry telling him that I feared the disease of the pancreas might be malignant, but there was a possibility of its being a chronic pancreatitis. On Nov. 5th I wrote a letter to this effect: "I am pleased to be able to tell you that your patient has improved very much, and the jaundice has nearly disappeared. I hope, therefore, that the tumour of the head of the pancreas may have been inflammatory and not malignant. At the time of operation it occurred to me that it was not quite hard enough for a malignant tumour, but under the circumstances I felt it my duty to give you my suspicions." From that time onwards recovery was uninterrupted, and the patient left the hospital with the wound closed within the month. In December 1899, the patient, at the kind suggestion of Dr. Berry, called to report himself to me. He looked perfectly healthy, and had gained over a stone in weight since his return home. He had neither pain nor tenderness, and he said that he felt as well as if he had never ailed anything. The scar was firm, the liver was normal, and there was not the slightest tenderness in the gall-bladder region.

CASE II.—*Chronic Pancreatitis; Cholecystotomy; Cure.*—The patient was a man aged 45 years, residing at Pickering. On 19th March, 1898, he was brought to see me by Mr. G. V. Robertson, of Pickering, the history being that he had been well up to twelve months before, when he began to have painful attacks at the pit of the stomach, ending in vomiting, but not followed by jaundice until an attack on 1st January, 1898, since which time he had been deeply and continuously jaundiced. He had also from that time onwards had ague-like attacks, and two days before seeing me he had had within twenty-four hours three of these seizures, each accompanied by pain. Within a twelvemonth he had lost 2 st. 8 lb. in weight. On examining him there was some swelling in the gall-bladder region, but no tenderness. The liver was a little enlarged, but the margins felt smooth. There was decided tenderness in the middle line just above the umbilicus, and on deep pressure the pain was considerable, and an indefinite fulness could be felt. The diagnosis of gall-stones in the common duct was made, and an operation was advised. The patient was operated on at a surgical home on March 30th, when the gall-bladder was found to be slightly distended and surrounded by adhesions to the pylorus, duodenum, colon, and omentum. No gall-stones could be discovered, but there was a swelling of the head and the first two inches of the pancreas, which, though nodular and irregular, was not very hard. This extended farther to the right than normal, so as to cover in the lower end of the common bile-duct. Cholecystotomy was performed. Within twenty-four hours of the operation nearly four pints of very offensive bile were discharged through the tube. A specimen was examined by the Clinical Research Association, and their report was as follows:—"The bile contains both staphylococci and streptococci, but no bacillus coli communis could be found either under the microscope or in the culture." Fearing that the disease might be malignant, and the patient being so extremely weak and ill I gave a poor prognosis, but in a few days I was able to write: "The patient is progressing very satisfactorily, though he is still profoundly weak. Bile has appeared in the motions, so that the obstruction is evidently overcome. The bowels have been moved naturally, and the patient is less deeply jaundiced and looking better generally." On April 5th I was able to report that he was taking food well, and that bile was passing freely in the motions. He had had no recurrence of the shivering attacks. The drainage was continued for fourteen days. On the 20th the patient returned home. The urine



was then free from bile, and the motions were assuming a natural colour; he was taking food well, gaining flesh, and looking better generally. I still, however, gave a guarded prognosis, though I said that I hoped that the tumour would prove to be inflammatory and not malignant. From that time onward his progress to recovery was extremely rapid. A report I had of his condition from Mr. Robertson a few months later said that he was perfectly well in every respect, and that he had fully regained his lost weight. I heard of this patient two years after his operation and he was still in perfectly good health.

These cases are given as characteristic examples out of twenty patients that I have operated on.

*Treatment.* — The course and treatment of chronic interstitial pancreatitis are exemplified in the cases related. Doubtless in some of these the manipulation of the indurated tumour detached calculi impacted in the duct, but in others the relief of tension as the result of draining the bile-ducts by cholecystotomy or cholecyst-enterostomy indirectly drained the pancreatic duct and thus led to a subsidence of the pancreatitis, then to an opening of the common duct by the relief of the tension, and so to a cure of the patient. The simulation of malignant disease of the head of the pancreas by chronic interstitial pancreatitis would make me hesitate to decline operation in any case of distended gall-bladder where the patient is in a condition to bear it, or even in any case of chronic jaundice without distension of the gall-bladder where the general health was deteriorating; as though it should be recognised that if the disease be really malignant very little good will be done, and life may even be shortened or only prolonged for a short time, yet if the disease prove to be chronic pancreatitis a real and permanent cure may be brought about. If a calculus be felt embedded in the head of the pancreas or impacted in the pancreatic duct it may be reached through the second part of the duodenum by laying open the papilla and exploring the duct, or by dividing the peritoneum passing between the duodenum and hepatic flexure of the colon, and then cutting through the overlying pancreas on the concretion. If the papilla be incised it does not require suture, and in the cases in which I have explored the ducts by the duodenal route there has been no serious hæmorrhage; the anterior duodenal opening requires closing by a mucous and a serous suture. Drainage of the right kidney pouch for twenty-four to forty-eight hours is advisable, though not always necessary, and this best done by a stab wound at the most dependent part.

For attacking the head of the pancreas or the pancreatic duct a vertical incision should be made through the right rectus and not in the middle line. In all cases of deep jaundice I prescribe chloride of calcium in 20 to 30 grain doses thrice daily for twenty-four or forty-eight hours before operation, and give it in enema for forty-eight hours afterwards in 60-grain doses thrice daily.

#### CYSTS OF PANCREAS

The term pancreatic cyst has been applied rather loosely so as to include all cysts occurring in the neighbourhood of the pancreas as well as true cysts of the pancreas itself. As was pointed out by Mr. Jordan Lloyd (*B. M. J.* 1892), many of these so-called pancreatic cysts result from an accumulation of fluid in the lesser peritoneal sac, this being especially true of tumours in the neighbourhood of the pancreas following on injury.

Cysts in the transverse meso-colon and in the omentum have also been frequently included under the term pancreatic cyst, so that it is impracticable to make a satisfactory study of the subject from many of the reported cases, or to rely on the accuracy of statistical evidence.



Though cysts may develop independently in the interlobular tissues of the pancreas, true pancreatic cysts are probably nearly always retention cysts, the result of obstruction in the ducts due to calculi or to changes in the walls of the ducts, the result of catarrhal ulceration or other forms of inflammation.

Senn pointed out (*Amer. Jour. of Med. Sci.* 1885) that a sudden complete obstruction of the pancreatic duct did not lead to cystic changes unless there was at the same time some disease of the gland itself.

As in the kidney, so probably in the pancreas, a sudden obstruction will lead to atrophy, but a gradually increasing obstruction to hydronephrosis in the kidney, and to cyst in the pancreas.

The relation of cysts of the pancreas to injury cannot be doubted, and though many of these cases are caused by effusion into the lesser peritoneal sac, some may be due to a laceration of the pancreas without rupture of the capsule, and then to accumulation of secretion and blood, which rapidly increases until a cyst is formed.

Hæmorrhagic cysts in the pancreas are probably nearly always traumatic, the exceptions being few; but pancreatic hæmorrhages do undoubtedly occur apart from injury in some unexplained way, perhaps due to reflex nerve influence, and a disruption of the soft pancreatic tissues may result in the formation of a cyst.

I have operated on a case of so-called false pancreatic cyst dependent on tubercular peritonitis, leading to effusion in the lesser peritoneal sac, this limited effusion being easily explained by the fact that two years previously the patient was apparently cured of tubercular peritonitis by suprapubic drainage, which led to the obliteration of the greater peritoneal sac, so that when there was a recurrence of disease, effusion could only occur in the unobliterated lesser cavity, leading to a swelling in the superior abdominal region.

I have also operated on a false pancreatic cyst due to traumatism, the injury being very slight and scarcely recognisable as an accident, namely, the sudden pressure of a knife-board on the epigastrium, caused by a slip of the foot when a butler was cleaning knives, leading him to slip forward, but not to fall against his board. At the operation a week later I found not only the lesser sac full of sanguineous fluid, but also the greater peritoneal cavity containing a quantity of the same fluid, which had passed through a small rent in the great omentum just below the stomach.

*Symptoms.*—In some cases of true pancreatic cyst, colicky symptoms like gall-stone attacks have preceded the formation of the cyst for months or years, but in others there have been no premonitory symptoms beyond slight digestive disturbances, until the tumour has been noticed, and then the symptoms have been those due to pressure on the stomach, and shortness of breath due to interference with the descent of the diaphragm.

Glycosuria and fat in the stools are quite exceptional symptoms, and when present are probably due to the whole of the pancreas being involved, which is unusual.

Loss of flesh may occur as the result of interference with digestion, and when pain is present, as the result of the painful attacks resembling cholelithiasis.

The tumour if sessile is fixed, but if the attachment be not very broad the top of the tumour may have a range of motion of 2 or 3 inches, and will then descend with respiration; it is situated behind the stomach, and on distending that organ with CO<sub>2</sub> the dulness will, as a rule, be quite hidden.

In one case on which I operated, a large cyst of the body and tail of



the pancreas extended well up under the left costal arch, and made the case resemble a cyst of the spleen. In another, the close apposition of the cyst to the left lobe of the liver, its moving with the liver on deep inspiration, the displacement of the stomach downwards, and the slow growth of the tumour, led to the suspicion of its being a cyst of the liver.

If the tumour be at the tail of the pancreas it may project into the loin and resemble a renal cyst, but, as a rule, bimanual palpation enables the kidney to be made out separately.

Tapping as a means of diagnosis or treatment should never be adopted, as it involves risk of perforation of viscera and of extravasation, and is attended with more danger than exploratory incision.

Pancreatic cysts may occur at any age. They have been noticed in infancy and in extreme old age, but they usually occur between twenty and forty.

The accompanying diagrams show the comparison between effusion into the lesser peritoneal sac and true cyst of the pancreas.

*Treatment.*—As regards treatment, pancreatic cysts present a direct antithesis to ovarian cysts, for in the latter drainage is disastrous, and removal the best treatment, whereas in the former removal is rarely justifiable, and drainage is usually curative. In only one case have I seen a pancreatic cyst that I felt could be safely removed, and in that case, the tumour being almost pedunculated, the removal was effected quite simply, and was followed by recovery.

The directions in which a pancreatic cyst may reach the surface and by which it may be approached are:—

- (1) Through the gastro-hepatic omentum "above the stomach."
- (2) Through the gastro-colic omentum "below the stomach."
- (3) Through the meso-colon "below the colon."
- (4) Through the loin behind the peritoneum.

Dr. Jules Boeckel (13th Internat. Congress) gives the following statistics of cases that he has collected from various sources. Of 144 operations for pancreatic cyst,—

Out of 99 cases treated by incision and immediate drainage there were 92 cures and 7 deaths.

Out of 16 cases treated by incision and drainage *à deux temps* there were 16 cures.

Out of 25 treated by removal, total or partial, there were 21 cures and 4 deaths.

#### MALIGNANT DISEASES OF THE PANCREAS

occur under the forms of both cancer and sarcoma, the former being much the more common; according to Segié, in the proportion of 63·5 to 1.

From the large number of cases of cancer of the pancreas that I have personally seen—considerably over 50—I should have thought it to be a fairly common disease; but Dr. Hale White (*Guy's Hosp. Rep.* liv.) gives only 30 cases of primary cancer and 1 of sarcoma among the 142 cases of pancreatic disease observed during fourteen years among 6708 examinations in the post-mortem room of Guy's Hospital. The table also shows 11 cases of secondary growth, and 13 in which the pancreas was involved by extension of malignant disease from adjoining organs; so that, taking all the cases together, it represents about one death from malignant disease of the pancreas out of every 200 patients that died in Guy's Hospital during fourteen years.

The disease, whether sarcoma or cancer, runs a very rapid course, and



an analysis of the Guy's series of cases shows that in no instance was life prolonged for more than eight months from the first appearance of symptoms, some patients dying within three months, and the sarcoma case within two months of the onset of symptoms.

Cancer of the pancreas is a disease of middle or advanced age. I have, however, seen it in a man of thirty-two, as well as in one at seventy-five. It is, nevertheless, unusual to find cancer of the pancreas before forty.

Sarcoma occurs earlier in life. In one case reported by Litten, the patient was only four years of age; a case of Dr. Goodhart's was twenty-eight. I operated on a man aged fifty-five for a large tumour presenting all the characters of a cyst of the pancreas, but on exploration it was found to be a soft sarcoma of extremely rapid growth, and the operation was therefore concluded as an exploration, which, if anything, appeared to benefit the patient for a time.

*Symptoms.*—The symptoms of cancer of the pancreas differ according to its site and the direction in which it progresses. If the body or tail only be involved there will be an absence of jaundice, but a steady wasting, with obscure epigastric pain and depression.

Pain is sometimes entirely absent, and the only signs are a steady loss of flesh and strength, and ultimately the appearance of a tumour behind the stomach; but at times the pain is severe and paroxysmal, then resembling gall-stones. Vomiting may be present or absent. Fat in the stools should be sought for, and when present is a valuable sign; but in many cases it is absent; in fact, its absence is the rule rather than the exception. Sugar in the urine is worth noting, as it is said to be present in about half the cases; but my experience is that glycosuria is not commonly present, and is, in fact, an exceptional symptom.

Among the cases coming under my observation I have found sugar present in three cases of cancer of the pancreas, but not in any of the acute conditions for which I have operated.

A hard, rounded, or nodular tumour, fixed deeply in the abdomen, just above the umbilicus, not markedly tender to pressure, not moving on respiration, giving a sense of communicated pulsation on palpation, and a bruit on auscultation, with stomach or bowel in front of it, and associated with rapid wasting, is likely to be a pancreatic tumour.

The growth only occasionally attains large enough proportions to be visible through the abdominal wall, and it may be so small as to be beyond detection, except as giving an obscure sense of resistance.

When the head of the pancreas is involved jaundice comes on and persists; a tumour is then readily felt, but this is the gall-bladder distended with mucus, and the pancreas itself cannot then ordinarily be palpated.

Persistent jaundice without pain, associated with tumour of the gall-bladder, is usually dependent on cancer of the head of the pancreas; and, in fact, whenever after forty there is chronic jaundice, associated with a distended gall-bladder, even with pain resembling gall-stone attacks, a suspicion of pancreatic cancer should be entertained.

Pressure on or involvement of the portal vein leads in the later stages to ascites, as does pressure on the vena cava to dropsy of the legs, and on the splenic vein to enlargement of the spleen.

The liver enlarges when the head of the pancreas is involved, owing to pressure on the common bile-duct and stagnation of bile in the liver.

The lymphatic glands and the liver and stomach become involved in the later stages. Bleeding from the gums, nose, and bowel, with petechiæ in the skin, nearly always appear in cases where jaundice is present. Infective



cholangitis, as shown by ague-like attacks, does occasionally occur in these cases, but much less frequently than when gall-stones are the cause of the obstruction in the bile-ducts.

*Diagnosis.*—The course and symptoms just related render a diagnosis feasible; but if in doubt with regard to the tumour, inflation of the stomach or colon will usually enable the disease to be distinguished from pyloric, bowel, liver, or gall-bladder tumour. The presence of lipuria or glycosuria and of fat in the stools afford valuable positive evidence, but their not being found is no proof of the absence of disease of the pancreas. In young subjects especially, but also in older patients, the symptoms of cancer of the pancreas are apt to be simulated by chronic interstitial pancreatitis; the surgeon should therefore not too hurriedly give up such cases as hopeless before surgical treatment has been tried.

*Treatment.*—Though I have seen a considerable number of cases of cancer of the pancreas, I have never yet found one where I considered excision feasible or worth a second thought; and although it is possible for a patient to live without the pancreas, as shown clinically and experimentally, I think it is scarcely likely that removal of cancer of this organ can ever be a frequent operation.

The facts established by Minkowski's experiments on dogs, and Senn's classical experiments, reported in 1886, show that not only life, but good health is possible where the pancreas has atrophied, or the duct has become permanently occluded. There is therefore no physiological reason why partial extirpation of the pancreas should not be performed for cancer if it can be discovered in time. But although Bilroth successfully removed a cancer of the pancreas in 1884, and Rugge removed with success a tumour believed to be retroperitoneal sarcoma, but which proved to be a primary carcinoma of the pancreas, the cases are exceptions, and do not establish the wisdom of the procedure.

Removal of the pancreas for primary cancer can be rarely a justifiable operation, unless it is taken at a very early stage, and unless the body or tail of the pancreas be the part affected; but under such circumstances the operation might be both feasible and justifiable.

If, however, it is rarely possible to remove cancer affecting the head of the pancreas, something may be done at times to relieve the jaundice that accompanies it, either by performing cholecystotomy, or by establishing a communication between the gall-bladder and bowel by a cholecyst-enterostomy.

I have operated on 16 cases of pancreatic cancer, and though the operations have as a rule been short and apparently favourable, the results have not been encouraging; these cases at the best are not promising, and even if recovery occurs, life is prolonged only for a short time.

M. Takayasu, of Osaka, Japan, collected 20 cases of cholecystotomy, all of which died shortly after operation, the longest survival being forty days, and 13 of cholecyst-enterostomy, all of which also only survived a short time. My own experience is more favourable; of 16 cases, 7 died soon after, and 9 recovered from operation and obtained relief from the jaundice. It seems to me that the only justification for operating is that there may have been an error in the diagnosis, and that the swelling of the head of the pancreas may be a chronic pancreatitis producing pressure on the bile-ducts, when, as I shall hope to show later, an operation may be absolutely curative.

Secondary malignant disease of the pancreas is not uncommonly found in both cancer and sarcoma, but in such cases other organs are also involved, and the pancreatic affection is beyond recognition, and has pathological rather than clinical interest.



The pancreas is frequently invaded by cancer owing to its proximity to other organs, such as the liver and stomach, the disease involving the pancreas by extension as the tumour increases; but in this case also the interest is merely pathological.

ULCERATION BY EXTENSION may give rise to pancreatitis ending in sloughing of the pancreas. In one case of this kind that I saw with Dr. Mercer of Bradford, in a man aged thirty-five, the abscess discharged into the stomach and led to vomiting of most offensive material, which appeared to be poisoning the patient, and made the atmosphere of the room almost too foetid to be borne by friends or nurses.

I performed gastro-enterostomy, although the patient was extremely ill, in order to give the ulcer a chance to heal, and that the offensive material might pass into the intestinal canal; the result was excellent, the patient making a good recovery, and being at the present time in excellent health.

Ulceration of the pancreas through extension of chronic ulcer of the stomach is not infrequently seen, and I have found specimens in several of the museums.

On three occasions I have operated and found extensive invasion of the pancreas by an ulcer in the posterior wall of the stomach. Under these circumstances the operation of gastro-enterostomy in two of the cases gave immediate and complete relief, and led to permanent recovery, the patients, both men of middle age, being now quite well. In the third case cancer had become grafted on the ulcer, and an attempt to remove the whole of the disease, though promising at first to be successful, ended in death the second week after operation.

PANCREATIC LITHIASIS.—Calculi in the pancreas are certainly not common, as proved by post-mortem records, by the paucity of specimens in the museums, and by clinical experience.

Dr. Hale White only found three examples at Guy's Hospital among 6708 post-mortem examinations during fourteen years.

M. Lancereaux, *Journal de méd. int.*, Feb. 1st, 1899, collected 40 cases, including one of his own; this is the largest number recorded, and probably includes all or nearly all the reported cases.

*Causes.*—Sedentary habits and over-feeding are predisposing causes, but atheroma of arteries, gall-stones, and chronic rheumatism are frequently associated with calculi in the pancreas. Obstruction of the ducts by the pressure of a tumour may be an exciting cause.

As stones in the pancreatic duct are usually associated with inflammation of the ducts or of the gland, it is highly probable that the true exciting cause is bacterial infection, as is now proved to be the chief cause of the formation of gall-stones.

Pancreatic calculi are whitish, and composed almost exclusively of calcium carbonate and phosphates. They are spherical, ovoid, or cylindrical in shape, but may be irregular, granular on the surface and branching, taking the shape of the ducts, in which they may be found free, but in some cases they are fixed in the substance of the gland. They may be single or very numerous, as many as 300 have been found in one case. They vary in size from that of a millet seed to that of a hazel nut.

There is a good example of multiple calculi in the pancreas in the Hunterian Museum, and in that specimen they are found in all parts of the gland, but the most common site is in the large duct, especially at the head of the gland.

*Symptoms and Signs.*—Paroxysmal pains at the epigastrium resembling gall-stone attacks, but associated with tenderness over the gall-bladder, and



generally unaccompanied by jaundice are usually the first symptoms. When inflammation occurs the attacks may be associated with ague-like seizures resembling infective cholangitis. As the calculi pass they interfere with the flow of bile from the papilla, and jaundice may be expected to occur, further obscuring the diagnosis.

The tenderness is in the middle line, and not just below the right costal margin, as in cholelithiasis, and the reflected pain is mid-scapular, and not under the right scapula, as in gall-bladder pain.

Glycosuria and fat in stools, rarely lipuria, may be present in advanced cases, but as these symptoms are dependent on pancreatic degeneration they are only found in advanced cases.

Lancereaux found persistent diabetes in 12 out of his 40 collected cases.

In one case recorded an epigastric tumour was felt; this was probably due to the formation of a cyst from obstruction of Wirsung's duct.

Acute pancreatitis may result, with suppuration, hæmorrhage, or even gangrene, but chronic interstitial pancreatitis followed by atrophy of the gland is more likely to follow.

*Prognosis.*—Recovery after the passage of the concretion is possible, but when the calculi are multiple serious secondary changes in the pancreas are usually present, and a chronic illness extending over months or years is probable, finally ending in diabetes. At any time acute pancreatitis may set in and produce a fatal termination.

*Treatment.*—Relief of the pain by sedatives, regulation of the bowels by aperients, careful attention to diet and the treatment of symptoms as they arise, practically sum up all that can be done by medical treatment, but if the diagnosis of pancreatic calculus can be made, surgical treatment offers the best chance of relief.

A vertical incision through the right rectus enables the pancreas to be thoroughly explored, and if a stone be felt, it may be got at either by dividing the peritoneum, passing from the duodenum to the colon, and then directly cut down on, or by opening the duodenum and incising the papilla common both to the pancreatic and common bile-duct.

If no calculus can be felt, but the paroxysmal character of the symptoms and swelling of the head of the pancreas lead to the suspicion of infection of the pancreatic ducts, a cholecystotomy with long-continued drainage of the bile-ducts may afford relief also to the pancreas, as in the cases described under the head of chronic pancreatitis.

**INJURY TO THE PANCREAS.**—Wounds of the pancreas generally entail speedy death, since from the deep situation of the organ such injuries are usually complicated by serious damage to other organs as well as by profuse hæmorrhage from the pancreas itself. The pancreas may be contused or torn across, when if death does not rapidly occur as the result of the injury, the blood effused in the neighbourhood may tend to form a cyst, as in one case that I saw in which the lesser peritoneal cavity had become filled with sanguineous effusion.

Lancereaux, in his work on the pancreas, described a case in which a bullet injured the pancreas, but here death appeared to be due to other injuries.

In one case occurring at Guy's Hospital, and described by Dr. Hale White, the patient was struck by the shaft of a cart, which injured the great omentum and ruptured the head of the pancreas. The patient survived a week, and at the post-mortem examination there was found extensive fat necrosis.

The only rational treatment is to cleanse the wound, to ligature obvious bleeding points, and then to plug the cavity with iodoform gauze. The



patient is usually suffering from such severe shock that any elaborate operation is out of the question, and though theoretically a search for the wounded vessels is advisable, practically the patient's condition prevents anything but a very rapid examination, and the adoption of such measures only as can be carried out rapidly.

**SYPHILIS OF THE PANCREAS.**—Syphilis of the pancreas is much commoner as a congenital than as an acquired disease.

In 1875, Birch-Hirschfeld described syphilitic disease of the pancreas in new-born children and infants, and Schlesinger (*Virchow's Archiv*, vol. cliv. Dec. 20th, 1898) has described six cases in which death ensued in every instance. He quotes that in congenital syphilis the pancreas is affected less frequently than the spleen, liver, bones, and lungs, but oftener than the thymus, heart, intestine, and other viscera. The pancreas may be attacked as early as the fifth month of uterine life. Peritoneal adhesions are commonly found around the pancreas. The head is always more affected than the tail, and on section it is abnormally firm so as to feel and cut like cartilage. The disease is thus really an interstitial diffuse pancreatitis. The interacinous tissue is much necrosed, and the vessels are affected with periarteritis and endarteritis. The ducts are but little affected except by the sclerotic changes of the tissues surrounding them. In congenital syphilis gummata are exceptional, whereas in the acquired variety they are characteristic.

**TUBERCLE OF THE PANCREAS.**—Pancreatic tuberculosis is extremely rare as a primary disease, though occasionally found in cases of general tuberculosis as well as in tubercular peritonitis. Tubercular disease of the pancreas may caseate and may break down into an abscess, which burrowing behind the peritoneum may tend to point in the loin or in the left iliac region. I have operated on a case of this kind in which the disease resembled a tubercular spinal abscess.

Lancereaux describes two forms: one composed of large caseous masses, the other of granular infiltrating tuberculosis. He quotes Kudrewetzki, who found the pancreas affected with tubercle fifteen times in 128 cases of tuberculosis.

Oser found even a greater proportion (44·4 per cent) of tubercular affections of the pancreas in tubercular children. He also describes one case in which a tubercular mass was excised from the pancreas.

Tubercular disease of the pancreas is of interest from a pathological rather than from a clinical point of view.

**Papilloma.** See TUMOURS.

**Paracentesis.**

See also ASPIRATION and ASCITES.

THIS term is applied to the operation of tapping one of the serous cavities for fluid, or, more rarely, gas. The operation is most frequently done for the removal of fluid from the peritoneal or pleural cavity. It may be carried out by a quick process, the use of a large trocar and canula, described under "Aspiration" in vol. i.; when it is desirable to promote the very gradual escape of the fluid, a much finer trocar, canula, and drainage-tube are employed (Southey's tubes).

This latter process has the merits of simplicity, it is less terrifying to the patient, and the tendency to syncope is reduced. Southey's apparatus consists of a very fine canula, about two inches long, perforated laterally by minute openings, and surmounted by a small shield. Over this shield

is passed one end of the rubber tube, the other end of which extends to the receiving basin under the patient's bed. It is then ready for use. The whole operation should be carried out with strict antiseptic precautions. After withdrawal of the needle the canula is fixed in position by strapping, and may be kept in for twelve or twenty-four hours. The use of an abdominal bandage will be found helpful, and it is better to gradually tighten the binder as the fluid escapes. A many-tailed bandage may be used. After withdrawal of the canula a collodion dressing is applied. Speaking generally, Southey's tubes are distinctly preferable to the aspirator for cases of ascites. An important preliminary measure is to see that the bladder is empty.

## Paralysis.

Paralysis is a symptom, not a disease. The present section deals with the so-called system lesions of the cord, and other diseases of the grey and white matter. Tabes dorsalis is treated separately in a later volume. Allied disorders are considered in the section on "Muscles and Myasthenia Gravis" in vol. viii., and in the section on "Spinal Cord" in a later volume. A general index of the nervous system will be found in vol. viii. p. 323.

### Paralysis

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*See also* SPINAL CORD.

THE term "paralysis" is that commonly employed to denote loss of motor power, irrespective of the amount of the defect, though the term "paresis" is sometimes applied to the lesser degrees of impairment of movement, while "paralysis" is reserved for the more pronounced defects.

The power of performing movements may be diminished or abolished in consequence of lesions of the sensori-motor region of the cortex of the



brain, of any part of the motor path from this region to the pons, medulla oblongata, and spinal cord, of the nuclei of motor cranial nerves, of the anterior horns of the spinal cord, of the peripheral nerves, and even of the muscles themselves. In addition to which there may be definite paralysis without its being possible to discover structural changes in any of the parts that have just been enumerated.

Paralysis may be acute or chronic, and the acute form may be of sudden or rapid onset, while the chronic form may be the legacy of a past acute affection, or its onset may have been gradual and its progress slow. In acute paralysis of sudden or rapid onset, when death does not result in consequence of the attack, there is commonly some tendency to improvement after the maximum effect of the lesion has been reached, and even if recovery does not result, there is no tendency for the paralysis to progress after it has become chronic. When it is of gradual onset and chronic from the outset, however, the tendency is for the paralysis to progress slowly but surely until a fatal termination is reached. Paralysis of sudden or rapid onset may result from affections of the brain or spinal cord, as may paralysis that is chronic from the outset. When the peripheral nerves are affected the most usual form of paralysis is that of rapid onset, though, notably in lesions of single nerves, the onset may be sudden, and in rare instances multiple affection of peripheral nerves is chronic from the outset. The almost invariable rule is that paralysis due to primary affection of the muscles is chronic in its onset and subsequent course.

The seat of the lesion which causes the paralysis determines certain distinguishing features in regard to its distribution. Thus, when the brain is affected the paralysis is usually limited to one-half of the body, including the face, trunk, and limbs, and the affection is then known as "hemiplegia"; but both sides of the body may be affected, including all four limbs, as is seen in cerebral "diplegia," or the paralysis may be limited to the lower extremities, and is then known as "paraplegia," which is, however, distinctive of a lesion of the spinal cord rather than of the brain. Even a single limb may be paralysed ("monoplegia") as the result of a cerebral lesion. Although "paraplegia" is the form of paralysis most characteristic of an affection of the spinal cord, "monoplegia" is also common. Both upper limbs may be affected while the lower limbs escape, and under rare circumstances the two limbs on the same side are involved, or even the upper limb on one side and the lower limb on the other. When lesions of the peripheral nerves cause paralysis the loss of power may be limited to the anatomical distribution of one or more nerves; but when there is a general affection of the peripheral nerves the paralysis is commonly paraplegic in distribution, or all four limbs may be weak. Less frequently the upper extremities suffer, while the lower extremities escape. The cranial nerves are rarely affected except when the paralysis is due to certain causes, notably the poison of diphtheria. Paralysis consequent on primary affection of the muscles usually involves all four limbs, especially their proximal segments, and the trunk; but, except in one form, the face escapes.

There are, however, some forms of paralysis in which the morbid process is not confined to one part of the nervous system, but attacks the brain, the spinal cord, and possibly also some peripheral nerves.

The seat of the lesion also determines the character of the paralysis which may be "spastic" or "flaccid" according to whether or not the muscles are in a state of spasm. Paralysis that is due to a cerebral lesion is usually spastic in type, and this is also the case when a lesion in the spinal cord is above the lumbar enlargement. There are, however, cases in



which with a complete transverse lesion above the lumbar enlargement flaccid instead of spastic paralysis results. Flaccid paralysis also occurs when the lesion is in the anterior horns of the spinal cord. So, too, the paralysis that results from lesions of peripheral nerves is of flaccid type, as is that due to primary affection of the muscles.

The state of nutrition of the paralysed muscles also depends on the seat of the lesion. When the lesion is in the brain, or interrupts some part of the motor path between the sensori-motor cortex and the nuclei of the motor cranial nerves, or the anterior horn cells of the spinal cord, there is no notable atrophy of the paralysed muscles; but when there is an affection of the nuclei of the motor cranial nerves, or of the anterior horns of the spinal cord, the paralysed muscles waste, a feature shared by all palsies due to lesions of peripheral nerves, or when there is primary affection of the muscles. Indeed, in the latter class of affection the degree of paralysis is proportional to the amount of atrophy of the muscles.

Although lesions in some situations do not cause atrophy of the paralysed muscles, it is noteworthy that a lesion of the central nervous system in the young, irrespective of its precise seat, is followed by retarded development, so that when these patients reach adult life, the maximum development of the paralysed parts is considerably less than that of corresponding regions on the opposite side of the body. This defect is not limited to the muscles, but is shared by the bones and other structures, and is well seen in those that have been attacked by acute anterior poliomyelitis (infantile paralysis), and in the subjects of infantile hemiplegia.

In cases of paralysis loss of motor power may be the only defect of movement, but in other cases this is associated with disordered movements which are spontaneous, or which are only evoked by voluntary effort. Athetosis or "mobile spasm," choreiform movements, and tremors of various kinds, may follow upon paralysis of cerebral origin, and are notably seen in infantile hemiplegia. In such cases there is always some return of voluntary power, and the disordered movement is not noticed for some months after the onset of the paralysis. Disordered movement is also seen in association with paralysis in diseases such as chorea, in which affection there may be weakness in addition to spontaneous movements; in disseminated sclerosis, in which intention tremor is such a prominent feature; in paralysis agitans, in which rhythmical tremor, commonly lessened or arrested by voluntary movement, accompanies the progressive weakness; in general paralysis of the insane, in which tremor of the tongue, face, and hands on voluntary movement is one of the characteristic features of the disease; and in the tremor that accompanies some forms of hysterical paralysis. So, too, in affections of the cerebellum the prominent symptom is inco-ordination of movement, but this may be accompanied by motor weakness; and in multiple peripheral neuritis, although motor paralysis is the chief feature, inco-ordination of movement may be added, and in some cases the latter symptom dominates the clinical picture.

Various deformities may result in paralysed parts, where spasm may bring about the distortion, or where they may be due to contracture and permanent shortening of muscles that happen to escape paralysis, or are less affected than their antagonists. Moreover, even when muscles are equally paralysed, posture may determine deformities by facilitating contracture of certain muscles, and thus some habitual position of a part may become permanent, unless altered by treatment.

Accounts of various forms of paralysis have already appeared in this and previous volumes of the *Encyclopædia Medica*; but a large number of



important affections of the kind have yet to be dealt with, most of which will be described in this article.

TABLE OF THE SPINAL SEGMENTS  
*Their Muscular and Sensory Supply and Levels*

Spinal Segment.	Corresponding spinous Process.	Muscles supplied.	Reflex		Sensory Distribution.
C 1		Small neck muscles	...	C 1	
C 2	C 1	Complexus. Splenius	...	C 2	Back of occiput
C 3		Depressors of hyoid bone	...	C 3	Anterior and posterior triangles of neck from ear to clavicle and scapula
C 4	C 2	Sternomastoid	...	C 4	Epaulette on shoulder, thorax from clavicle to three inches below
C 5	C 3	Lev. ang. scap., scaleni. Diaphragm	...	C 5	Deltoid region and outer border of arm narrowing to point just above ball of thumb (radial border)
C 6	C 4	Spinati, deltoid, biceps, brachialis anticus, sup. longus, pectoralis major (clavicular head), servatus magnus	Radius jerk	C 6	Deltoid region and outer border of arm narrowing to point just above ball of thumb (radial border)
C 7	C 5	Pectoralis major (pectoral head), subscapularis, teres major, latissimus dorsi	...	C 7	Strip down front and back of arm (inside C 5), radial border of hand, thumb, index and radial half of middle finger
C 8	C 6	Triceps, flexors of wrist, extensors of wrist	Triceps jerk	C 8	Strip down front and back of arm, contiguous halves of middle and ring fingers
D 1	C 7	Flexors of digits, extensors of digits	...	D 1	Strip down front and back of arm, inner half of ring finger, little finger, ulnar border of hand
D 2-3	D 1	Intrinsic muscles of the hand	...	D 2-3	Post-axial border of forearm from three inches above elbow to pisiform bone
D 4	D 2	Dilators of the pupil	Pupil dilatation to shade	D 4	Post-axial border of forearm above D 1
D 6	D 4	...	Epigastric	D 5	Level of nipple
D 9	D 6	Inter-costales (D 1 to D 11), erector spinae (C 3 to S 3)	Abdominal	D 6	Level of xiphisternal junction
L 1	D 10	...	...	D 9	Level of umbilicus
L 2	D 11	Abdominal muscles (D 6 to L 1)	...	L 1	Level of Poupart's ligament
L 3		...	Cremasteric	L 2	Upper and outer side of thigh and over Scarpa's triangle
L 4		Cremaster	Knee-jerk	L 3	All thigh except narrow vertical strip on post surface (S 2). Ceasing about an inch above knee
L 5		Ilio psoas, adductors of hip. Sartorius	...	L 4	Knee except behind. Inner aspect of leg to ankle
		Extensor quadriceps cruris. Abductors of hip	...	L 5	Outer aspect of leg, dorsum of foot
		Flexors of knee	...		

TABLE OF THE SPINAL SEGMENTS.—*Continued.*

Spinal Segment	Corresponding spinous Process.	Muscles supplied.	Reflex.		Sensory Distribution.
S 1	D 12	Calf muscles	Foot clonus	S 1	Dorsal aspect of toes, instep, sole of foot, heel and two strips enclosing S 2 on back of calf reaching almost to knee
S 2		... Glutei, peronei, ant. tibial muscles, intrinsic muscles of foot	Plantar reflex ...	S 2	Strip along back of lower extremity from two inches above heel to above middle of buttock
S 3		Perineal muscles	Anal reflex		Perinæum, genitals, adductor region of thigh
S 4					
S 5	L 1				
Cocc.					Sacral region

## I. SPASTIC PARALYSIS

SPASTIC PARAPLEGIA.—The essential characteristic of this symptom group which may be produced by the most varied pathological processes, having their site in many different parts of the central nervous system, is bilaterally distributed paralysis, associated with increased muscular tone (rigidity).

*Muscle Tonus.*—Every healthy muscle in the living state exists normally in a state of slight contraction or tonus. In slight degree this tonus is an expression of the life of the muscle apart from other influences; in much greater degree it is dependent upon the nerve cells directly governing the muscles (lower motor neurons). This factor in muscle tonus is produced reflexly. If attention be called to any part of the body, sensations coming from that part will reach consciousness. Such unfelt sensory stimuli are constantly impinging upon the spinal segments, and find their expression upon the motor side in a continuous outflow of energy distributed to the muscles and producing a condition of continuous slight contraction—the spinal element of muscle tonus. From this reflex origin it is obvious that a lesion of the sensory nerve at any part of its course from the periphery to the spinal segment, or a lesion of the spinal segment itself, or a lesion of the motor nerve at any part of its course, will abolish the muscle tonus. In other words, muscle tone is dependent upon the integrity of the reflex arc. Further, the spinal segments themselves are presided over by higher parts of the nervous system which regulate the activity of the response of the spinal segment to incitations coming from the periphery. This higher mechanism has, on the one hand, an inhibitory influence upon the spinal segment, and, on the other hand, an augmentor influence. The inhibitor influence is cerebral in origin, and is distributed to the spinal segments by the fibres of the pyramidal tract. If this influence be removed, as in a lesion of the pyramidal system, the spinal segment reacts to the constant afferent impressions unrestrainedly, and its physiological capacity for such action slowly increases. Consequently the expression of the constant activity of the spinal segment, the muscle tone, increases, and rigidity is produced. The augmentor influence arises in some part of the central nervous system, below the midbrain and above the medulla. Its path in the spinal cord is not definitely known, but this does not cross in the medulla or spinal cord. If in the cat the cerebral hemispheres be removed, there ensues at once



intense extensor rigidity of all four limbs—decerebrate rigidity. Semi-section of the spinal cord or medulla abolishes this rigidity on the side of semisection below the point of section. Decerebrate rigidity, therefore, must be due to incitations having their seat in the ponto-cerebellar region.

In lower animals (cat and dog) the spinal segments possess sufficient autonomy not only to maintain muscular tone, but to produce rigidity when severed from all connection with the higher parts of the nervous system. In the higher apes this autonomy is much feebler, and in man the spinal segment cannot *per se* produce any degree of muscular tone. From this the most important clinical fact arises, that a complete transverse lesion of the spinal cord produces completely flaccid paralysis with loss of deep reflexes, while an incomplete lesion produces spastic paralysis, for the augmentor influence still maintains a hold upon the spinal segments below the lesion. Clinically the terms “hypertonus” and “hypotonus” are used for increase or decrease respectively in the muscle tone.

*Myotatic Irritability (Tendon Reflexes).*—These phenomena are responses of muscles in a state of tonus to mechanical stimulation applied in a certain way. They are increased, diminished, or lost in proportion as the muscular tone is increased or diminished.

They are only reflex in the sense that the muscle tone necessary for their production is dependent upon the integrity of the reflex arc.

The above physiological considerations are absolutely necessary for the clinical study of spastic paraplegia. We have seen that in man the muscle tone depends upon the integrity of two entirely distinct nervous mechanisms—firstly, the reflex arc consisting of sensory nerve, spinal segment, and motor nerve; and, secondly, the integrity of the higher parts of the nervous system and the tracts connecting these with the spinal segments.

Classification of the diseases in which spastic paraplegia is a characteristic symptom.

A. Diseases of the nervous system in which spastic paraplegia appears as a primary symptom.

(1) Diseases confined to the pyramidal system—

Primary lateral sclerosis.  
Familial spastic paraplegia.  
Cerebral diplegia.

(2) Diseases affecting the pyramidal system and other parts of the nervous system—

Disseminated sclerosis.  
Ataxic paraplegia.  
Amyotrophic lateral sclerosis.  
Hereditary cerebellar ataxy of Marie.  
Syringomyelia.  
Spina bifida.  
General paralysis of the insane (certain cases).  
Syphilitic paraplegia.

(3) Diseases of probable toxic origin—

Pellagra.  
Lathyrism.  
Subacute combined degeneration.

B. Diseases of the nervous system in which spastic paraplegia results as a secondary process—

Acute myelitis.  
Hæmatomyelia.  
Spinal meningitis.

C. Injury to the nervous system—

1. By gradual pressure.  
Tumours of the spinal cord or meninges.  
Tumours arising in or secondarily involving the vertebral canal, aneurysm.  
Spinal caries.  
Pachymeningitis.
2. By sudden violence.  
Fractured dislocation of the spine.  
Gunshot and other wounds of the spinal cord.

From this it will be seen that where spastic paraplegia exists there must be some loss of the influence usually exerted by the pyramidal system upon the spinal segments either from degeneration of the nerve elements or interruption of the spinal fibres of that system. The spinal segments must be themselves capable of carrying on their reflex functions, and this last condition requires that there is still some connection of the spinal segments with the higher parts of the nervous system.

The lesions producing spastic paraplegia may be situated at any part of the pyramidal path from the cortex of the Rolandic area to the upper limit of the lumbar enlargement of the spinal cord. In the cerebral hemispheres such lesions must of necessity be bilateral to produce paraplegia, and the resulting conditions are distinguished by the term "diplegia," which indicates a lesion of cerebral location. In the base of the brain, pons, and medulla oblongata the pyramidal tracts are less widely separated, and may be simultaneously involved by a lesion of considerable size, but it is in the spinal cord, where these tracts are close together, that the pathological conditions which produce spastic paraplegia are most often situated. In primary spastic paraplegia and in amyotrophic lateral sclerosis, however, the whole length of the pyramidal tract is affected by a degenerative process.

The nature of the lesions is most varied, and may be either a degenerative process, primary in the nerve elements, a vascular lesion, a new growth, or injury to the nervous system by gradual pressure or sudden violence.

*Clinical Aspect.*—The essential features of spastic paraplegia, loss of power, with rigidity and increase of the deep reflexes, are rarely the sole conditions present, and it is only in primary degenerative conditions of the pyramidal system that other symptoms are entirely absent. For a time, however, such a simple picture may be present in the early stages of many of the above-mentioned diseases, especially in disseminated sclerosis and in compression of the spinal cord.

The majority of lesions affect other systems besides the pyramidal, and signs of involvement of the sensory tracts, and perhaps of the gray matter, occur with or are added to those of simple spastic paraplegia. The pyramidal system appears to be less resistant to the effects of pressure, sclerosis, and vascular lesions of slow occurrence than the other structures of the spinal cord, and perhaps this feature may be connected with its later appearance in evolution. Consequently a derogatory influence, such as progressive pressure upon the spinal cord or local vascular degeneration, will abrogate the functions of the pyramidal tracts first and most, though all the elements of the marrow may be exposed to the harmful influence.



When recovery takes place again the pyramidal tracts are the last structures to regain their physiological activity. A lesion gradual in onset and slowly progressive, which affects the whole spinal cord at one point, such, for instance, as a collection of tubercular material, or a tumour pressing upon the spinal cord, will affect the various structures, and produce symptoms in an order which is for the most part constant. Firstly, the pyramidal tracts are affected, and loss of power, with rigidity and increase of the deep reflexes, appears. The tracts subserving the muscular sense and sense of passive position are the next to fail, and ataxy, with loss of sense of position, results. The paths by which sphincters are voluntarily controlled are deranged later, and retention of urine and constipation with involuntary sphincter action occur. Last of all, the undefined tracts of general sensibility are interrupted, and anæsthesia of common sensibility is produced. There is still one further stage—that where all the conducting functions of the spinal cord are lost at the place of injury, the connection of the higher parts of the nervous system with that part of the spinal cord below the seat of the lesion is cut, and muscular tonus in the paraplegic area is lost. The previous aspect of spasticity with increased deep reflexes changes at once to flaccid paraplegia with loss of the deep reflexes. The supervention of a stage of flaccidity in spastic paraplegia due to pressure is of the gravest significance, for recovery is possible, indeed not improbable, in many cases where motor paralysis is absolute and anæsthesia complete, even though this condition may have existed for weeks or months, if there is still spasticity, but when the flaccid stage has set in, complete recovery becomes impossible after a few days.

The symptoms disappear in the reverse order when recovery takes place, except that sphincter trouble is apt to remain longest. For instance, a case in which a tumour pressed upon the mid-dorsal cord presented the above sequence of symptoms during a period of two months. Four days after the paralysis and anæsthesia had become complete, flaccidity with loss of the deep reflexes set in. Three days later the tumour was removed. After the operation the knee-jerks returned upon the third day; there was marked spasticity on the seventh day. The first sign of returning sensibility was noticed on the seventeenth day, the first voluntary movement on the thirty-fourth day, and some sphincter trouble lasted for three months. This patient returned to farm labour seven months after the operation with no abnormal physical signs.

In addition to these symptoms, the lesion producing spastic paraplegia may give rise to local manifestations of great importance in determining the location and pathological nature of the disease. The anterior horns of the gray matter or the anterior roots may be involved and local muscular wasting and paralysis, with changes of the electrical excitability of the muscles, may result in the region immediately above the paraplegic zone. The posterior roots and their prolongations may be affected, and anæsthesia of nerve root distribution, areas of hyperæsthesia, pains radiating along the course of certain nerves, girdle sensation, and occasionally herpes zoster may result. If the lesion be situated in the cervical region, or implicate the first thoracic nerve root, changes in the pupil, in the width of the palpebral aperture, and vascular disturbances of the face may result.

When spastic paraplegia results from widely-spread disease of the nervous system, as in disseminated sclerosis, general paralysis, and syringomyelia, it is not as a rule sharply limited, often not of severe degree, and is sooner or later accompanied by some of the multitudinous symptoms of these diseases.



Acute processes affecting the spinal cord locally, such as acute myelitis, hæmatomyelia, fractured dislocation of the spine, and gunshot and punctured wounds, except in cases of the slightest severity, produce at first a condition of flaccid paralysis with loss of the deep reflexes, from which in the course of a few hours or days spastic paraplegia develops. Such initial flaccidity may be explained by the phenomenon of shock as a condition of inhibition, or it may be presumed that a temporary complete physiological interruption of all the conducting elements of the spinal cord occurs without complete anatomical destruction as a result of the lesion, and that such elements as retain their anatomical continuity speedily regain their function.

When, however, complete anatomical destruction occurs, the paraplegia remains flaccid and the deep reflexes are absent in the paraplegic region. It must, however, be remarked that if after a total lesion of the cord a patient live many months, he may ultimately regain the deep reflexes, and some slight signs of spasticity may return. It is possible that this rare occurrence may be due either to the regeneration of fibres reproducing continuity of the severed cord, or to the resumption of such autonomy by the gray matter of the distal part of the spinal cord as exists in the lower animals.

*Motor System.*—Where spastic paraplegia is of gradual onset, as in the majority of cases, loss of power shows itself, firstly, in the periphery of the limbs, and as a general rule the extensor muscles, abductor muscles, and external rotator muscles fail before the flexor, adductor, and internal rotator muscles. The first difficulty noticed by a patient is usually a tendency for the toes to catch the ground and to become inverted in walking, and from this the boots are worn down at the toes and on the outer side in a peculiar fashion.

At a later stage the gait becomes characteristic: the patient walks upon the toes with the feet inturned, the knees approximated and slightly flexed, and the thighs adducted, internally rotated, and slightly flexed, so that the body is carried forward. The difficulty in performing the abduction at the hip necessary for projection of the leg in walking is often aided by tilting of the pelvis alternately to the right and left with each step.

In the upper extremities, when these are affected, the wrist and fingers tend to be semi-flexed, the elbow flexed, with the forearm carried across the chest, and the arm adducted to the side.

Muscular rigidity appears *pari passu* with the loss of power, sometimes preponderatingly, so that patients attribute their voluntary incapacity to the stiffness. Slowness on effort and resistance to passive movements are its signs, and these are later added to by the occurrence of contractures and the production of characteristic positions of the limbs, which in time are rendered permanent by alterations in the bony and ligamentous structures. The common position of the foot is known as *pes cavus*. In this deformity the foot is dropped, and often inverted, the plantar arch is increased, and the toes are hyperextended, so that they frequently form a right angle with the dorsum of the foot. The foot, viewed from its inner side, shows a Z-shaped border. The knee is generally rigid in the extended position, the rigidity suddenly lessening directly the knee is bent by passive movement—clasp-knife rigidity. Occasionally the rigidity persists equally in all the stages of passive flexion. In late stages, however, it is the rule for flexor spasm to preponderate at the knee and contracture of the hamstrings to occur. At the hip flexor and adductor spasm preponderates, and frequently contracture occurs.



Where spastic paraplegia exists in young children it is not uncommon for scoliosis or lordosis to occur.

The position of the upper extremity is usually one of flexion and adduction at all joints. Only in the rarest cases is a "manus cavus" comparable to "pes cavus" met with. The wrist is then flexed and the fingers hyperextended at all joints.

Contracture is a structural permanent shortening of the muscle. It is associated with defects of the pyramidal system, and is not to be explained by either muscular paralysis and unopposed action of antagonistic muscles, nor as a result of hypertonus, for both the latter may exist for years without contracture, and, conversely, contracture regularly occurs where there is neither rigidity nor an appreciable degree of motor paralysis in Friedreich's disease, in which, however, the pyramidal tract defect is present.

The increase of spasm by peripheral stimuli is a common feature of severe degrees of spastic paraplegia. The causes exciting these "reflex spasms" are often slight, a draught of air, a slight movement of the bed-clothing, or the touch of a cold finger, being sufficient to produce the most powerful spasms. In the lower extremities these are most frequently flexor in type. Such reflex flexor spasms appearing in the course of spastic paraplegia are of the gravest significance to the patient, for his disease, perhaps hitherto quite painless, becomes with their manifestation a most painful one; to the physician, for they yield to no treatment and mark an irrecoverable stage of disease (Gowers); to the nurse, for the sacral bed sore is almost inevitable, since the friction from the repeated sudden spasms cannot be avoided.

In the abdominal muscles, etc., spasm, either constant or reflex, may very considerably embarrass respiration.

There is frequently some general wasting of the muscles in spastic paraplegia of long duration; but where reflex spasms occur it is said that the muscles involved may be even hypertrophied. The electrical excitability is unaltered, the slight raising or lowering of Faradic excitability commonly met with being due to altered conditions of the conductivity of the skin and subcutaneous tissues resulting from the paraplegia.

*Sensory Manifestations.*—Subjective sensory phenomena occur frequently in spastic paraplegia, due to local disease of the spinal cord. Dull fixed pain in the back and a sense of constriction encircling the body, or of distension—the so-called girdle sensations—are common in both intra and extra-medullary lesions. Severe and sudden pains radiating over the areas supplied by the nerve roots indicate implication of the nerve roots, and are in favour of a lesion involving the meninges. These are not produced by movement of the spinal column, though often increased by such movement. Sharp pain actually produced by movement is common where the paraplegia is secondary to disease of the vertebral column.

*Objective Sensory Manifestations.*—Anæsthesia is distributed segmentally upon the body in local lesions (*vide* p. 114). As the result of pressure and of vascular disease it is usual for pain to be lost earliest, then temperature, then the muscle sense, and lastly touch. In lesions affecting the central parts of the cord especially, such as syringomyelia, hæmatomyelia, and central myelitis, sensibility to pain, heat, and cold are lost first, and often out of all proportion to the tactual impairment. Even in these cases, however, it is the rule for slight tactual alteration to coexist with and over the same area as the analgesia.

Trophic and vaso-motor disturbances are not conspicuous, though



frequently branny desquamation, thinning of the skin, depilation, and slight vaso-motor palsy may be present.

When the lesion is situated above the thoracic cord and the vaso and visceromotor paths are interrupted, abdominal distension and great lowering of the blood pressure may result and are of the gravest importance.

The typical condition of the sphincters in spastic paraplegia is one of reflex incontinence, but this condition is by no means invariable. Retention, with overflow, is not uncommon, and in a few late cases complete incontinence with patulous sphincters may occur, though the lumbar enlargement of the spinal cord is quite intact. Sexual power is lost *pari passu* with penile sensibility.

*Reflex Action.*—Superficial reflex action is increased below the lesion in partial lesions of the spinal cord and diminished in total transverse lesions. It is lost in the area actually supplied by that part of the spinal cord involved in the lesion.

Whenever the pyramidal tract is damaged the reflex obtained from the sole of the corresponding foot is altered, the response consisting of extension of the toes, especially of the great toe. The normal response consists of flexion of the toes, least marked in the great toe. This phenomenon is one of the most important signs in the differential diagnosis of lesions of the pyramidal tract. It is absolutely essential, however, to exclude voluntary movement and irregular reflex movement before a reflex response from the sole of the foot is finally determined to be of the extensor type, and, except in the hands of a practised observer, this is not always a simple matter.

*Tendon Reflexes.*—As has been pointed out above, the tendon reflexes are increased below the lesion except where the lesion of the spinal cord is total at a given level.

The tendon reflexes are lost in the area supplied by that part of the spinal cord actually involved in the lesion.

The associated phenomenon of clonus may be obtained in any muscle of the paraplegic region if suitable tension and mechanical stimulus be applied. General clonus of all the muscles, the so-called epileptoid spasm, is readily obtainable in most of the severe cases by jarring the whole limb. It may be set up by a reflex spasm, and this has been called spontaneous clonus.

*Diagnosis.*—(The diagnosis of paraplegia of cerebral origin will be found under the headings Double Hemiplegia, Diplegia, Cerebral Tumour, Pontine Lesions, etc.)

In spastic paraplegia of spinal origin it must be first determined whether the lesion is local, diffuse, or systemic.

The presence of signs and symptoms of segmental distribution sharply defined, such as girdle sensation, root pains, sharp lines of anæsthesia, and abrupt delimitation of muscular paralysis, characterise local lesions. Local signs in several widely-separated regions, such, for instance, as this combination which occurred in a case of disseminate sclerosis—slight spastic paraplegia of the legs, right trigeminal anæsthesia and paralysis, left third nerve paralysis, nystagmus, and optic atrophy—suggest diffuse or multiple lesions. The absence of all local signs for months or years allows a diagnosis of systemic degeneration. The diagnosis is, moreover, in all cases aided by the clinical history.

If a local lesion is present, (1) the situation, (2) the segmental level, and (3) the probable pathological nature of it must be determined.

(1) *Situation.*—The lesion may be situated in the substance of the cord, in connection with the meninges, or in connection with the vertebræ. The



diagnostic points are here, the occurrence of pain, the implication of nerve roots, and the presence of local signs of vertebral disease.

In intramedullary lesions pain is never a conspicuous symptom (the fixed pain of syringomyelia being excluded). Severe pains shooting along the nerve root distributions, not caused by movement, though perhaps increased thereby, are characteristic of lesions implicating the meninges, and such pains usually precede the appearance of paraplegia for a short time. Local muscular atrophy of peculiar distribution, from implication of the anterior roots, also frequently occurs.

Local signs of disease of the bone of the vertebral column, deformity, tenderness, pain on movement and on jarring the spine, and the presence of abscess, aneurysm, or cancer, suggest a lesion of the spinal column.

Girdle sensations may occur with each situation of the disease, but are more common in intramedullary disease.

*Determination of the Level of the Lesion.*—The upper limit of anæsthesia upon the trunk and limbs serves as an almost infallible guide to segmental location. Sensation must be tested in perpendicular lines upon the trunk, and both perpendicular and transverse lines upon the limbs, the slightest change noticed by the patient in passing from an area where sensation is perfect to where it is imperfect must be taken as this upper limit. No part of the body should escape at least rough testing, and tactual, painful, and thermal stimuli should be applied. It is not clear whether the upper limits of anæsthesia found in local disease of the spinal cord correspond with the limits of segmental sensory supply or nerve root sensory supply, but this doubt does not affect their localising value. (See Fig., p. 114).

The upper limit of muscles involved in the paralysis is the next most applicable sign in localisation, but it is not so exact nor so easy to investigate as is the anæsthesia. When the lesion is situated on or above the fourth cervical segment there is paralysis of the diaphragm. From the fourth cervical to the first thoracic segments the involvement of certain groups of the muscles of the upper extremity will indicate damage to each segment. The pupillary phenomenon, etc., also indicates damage above or in the first thoracic segment.

In the upper half of the thoracic region, since the only muscles supplied by this region of the cord are the intercostal and spinal muscles, narrow limitation of paralysis is not apparent. It must here be mentioned that the expansion movement of the five lower ribs is caused by the diaphragm, and exists where there is complete intercostal paralysis. From this it happens that intercostal paralysis is frequently overlooked. In the lower thoracic region, however, the most exact localisation is often possible by means of a sign first used by C. E. Beever. The patient is examined in the supine position with the anterior wall of the abdomen bare, and is directed to make the attempt to sit up. As he does so the junction of the contracting with the non-contracting muscle often becomes plainly visible as a retraction ring. Where the subcutaneous tissue of the abdominal wall is too abundant for the usual perception of this phenomenon, it may be felt with the hand. Further, the umbilicus, which in the normal state does not alter its position on the attempt to sit up, shifts upwards during such attempt if there is a lesion in the lower thoracic region.

The state of the superficial and tendon reflexes is only a guide in that the bilateral absence of one of them indicates that the segment subserving it is destroyed.

The zone of hyperæsthesia sometimes occurring at the upper limit of anæsthesia is of considerable localising value, but it is only rarely present.



Girdle sensations are also valuable, as are root pains, but sometimes they do not indicate the upper limit of disease.

Far more important than the last are local signs of disease in the vertebral column, deformity, and tenderness; but these signs are often too diffuse for exact localisation, and should never be relied upon alone to indicate the level of the lesion.

The determination of the pathological nature of the disease depends so much upon the history, clinical aspect, and course of each case, that few general statements of value can be made. From the mode of onset it may be broadly stated that if this be sudden the lesion is a hæmorrhage. If it be acute (1-3 days), myelitis (thrombosis) is most often responsible. If subacute (1-3 months), pressure upon the spinal cord is most likely. Toxic processes are chronic, and degenerative processes still more chronic. To each of these statements, however, exceptions are not rare. If pain be a prominent and early symptom, a pressure lesion or pachymeningitis may be safely inferred, but the converse does not hold good; as an instance, the paraplegia of spinal caries may be unassociated with pain. Pain is as a rule absent in vascular lesions, including myelitis, and in degenerative lesions.

The differential diagnosis of the diseases associated with spastic paraplegia will be found under the titles on p. 104.

It is useful to bear in mind that a case of spastic paraplegia which does not present signs at once furnishing a diagnosis is most often due to acute myelitis if the onset be acute, to spinal caries if subacute, and to disseminated sclerosis if chronic.

**PRIMARY LATERAL SCLEROSIS.**—There has been much discussion as to whether a primary system disease limited to the pyramidal tracts occurs affecting the lower end of the upper neuron pyramidal fibre in a way comparable to the affection of the lower neuron (peripheral nerve) in multiple neuritis. A condition nearly approaching to this has been found in certain cases of general paralysis of the insane, but it is here not an uncomplicated process. No convincing pathological records are forthcoming upon which a pathological entity of primary acquired lateral tract degeneration can be based. It is said, however, in explanation, that this disease rarely destroys life. Notwithstanding, however, the absence of satisfactory pathological data, there seems to be a group of cases to which the name primary lateral sclerosis may appropriately be given.

*Causation.*—As in other chronic spinal cord diseases occasional neuro-pathic heredity can be traced. Males and females are affected in about equal numbers. The symptoms usually appear in the third and fourth decades of life.

Primary spastic paraplegia sometimes follows syphilis in a way to suggest a causal relation, but this antecedent is not frequent. The onset of symptoms sometimes follows injury to the spine, exposure to wet, cold, over-exertion, and general illness.

*Symptoms.*—Gradually increasing weakness of the legs is the earliest symptom to appear. The patient finds that he becomes tired more easily than before, and the legs feel heavy. Often one leg is affected before the other. The tendency to spasm is at first noticeable as slight stiffness of the legs in rising in the morning, and this gradually increases in degree as power lessens.

Power is lost earliest in the anterior tibial group of muscles. The legs are rigid in extension and the clasp-knife phenomenon is well marked. Rarely short attacks of flexor spasm occur chiefly at night. It is only in rare instances that the arms are affected.



The clinical picture is that of spastic paraplegia, and for its details the reader is referred to the general description above given.

No sensory disorders are present with the exception of dull pain in the back. The sphincters are unaffected, and no girdle sensation is present. Cranial nerves and special senses are not affected.

*Diagnosis.*—There are so many diseases of the spinal cord of which the aspect at some period of their course resembles pure lateral sclerosis, that a correct diagnosis can only be arrived at by a process of exclusion. This disease has no pathognomonic symptoms, its characteristic being the absence of signs pointing to lesions of parts of the central nervous system other than the pyramidal tracts. It is clear that only by watching the progress of a case over a long period can a probable diagnosis be arrived at. A case exactly answering to the above description of simple spastic paraplegia may develop the fixed pupil, tremors, and mental disturbance of general paralysis; the appearance of nystagmus and ocular palsies, intention tremor and sphincter trouble, may necessitate that the diagnosis is changed to disseminated sclerosis; muscular wasting in the small muscles of the hand marks the case one of amyotrophic lateral sclerosis; and the appearance of local signs in the vertebral column may spell spinal caries. For this reason, in a case seen soon after the onset of the symptoms, the diagnosis of primary spastic paraplegia is one that must be made tentatively to serve until the appearance of some other symptom labels the disease accurately.

If after years such symptoms fail to appear, the diagnosis may be considered as finally correct. The majority of cases with this clinical aspect in young adults without a history of antecedent syphilis are cases of disseminated sclerosis, and it is astonishing in this disease how long the more classic symptoms, such as nystagmus, ocular palsies, altered speech, tremor, etc., may remain absent.

*Prognosis.*—In primary lateral sclerosis there is some prospect of arrest and even of improvement if the disease has not reached an advanced stage. Actual recovery is rare, but is said to have occurred. When the spastic state is developed it is rare for treatment to produce more than arrest. There is not at present any guide to the prognosis of any individual case, besides its duration and degree, except its observed tendency especially under treatment.

*Treatment.*—Drugs have little influence upon this disease. Arsenic and mercury in small doses; iron, quinine, and nux vomica have proved useful. The last of these must be used with caution, as it tends to increase the spasm. Massage and passive movements are often of signal service. Electricity in any form seems to do harm by exciting reflex spasm. Rest in bed seems to have ushered in improvement in many cases, but it should never be absolute, as such patients kept strictly in bed may lose the power they previously had from disuse, and are sure to attribute the deterioration to the confinement, perhaps not altogether incorrectly. Fraenckel's exercises often do good, and they, at the same time, occupy and interest the patient.

**FAMILIAL SPASTIC PARAPLEGIA.**—The occurrence of primary lateral sclerosis in several members of the same family was first noticed by Erb and Strümpell in 1888. The disease is a rare one and affects males and females equally, and may be directly transmitted to children through either parent. Several members of each generation as a rule suffer, and those members of an affected family who are said to be healthy, if carefully examined, are often found to present slight signs of spasticity, such as pes cavus, foot clonus, and slightly spastic gait, though they may never reach a degree



sufficient to interfere with comfort and daily labour. Other neuropathic tendencies sometimes are present in the family, such as insanity.

The symptoms become obtrusive between the ages of puberty and thirty years. Clumsiness in walking and stiffness of the legs are first noticed, subsequently the movements of the hands become slow and awkward, and general slowly progressive spastic paraplegia results. In some cases spastic bulbar symptoms, dysphagia, dysarthria, etc., have arisen. There is no alteration of sensibility, and the sphincters are unaffected. Pes cavus is invariably present. A slowly progressive mental hebitude is common, and the disposition is placid and good tempered.

In some cases progressive atrophy is present. After many years the patients may become bed-ridden, or the condition may cease to progress in a slight or severe stage of spastic paraplegia.

*Pathology.*—This disease seems to depend upon an inherent weakness of the pyramidal cells of the cortex cerebri, which after a longer or shorter tenure of vitality undergo degeneration.

*Treatment.*—No special treatment is known to influence the disease, but the measures recommended for the treatment of primary lateral sclerosis may be adopted.

**SYPHILITIC PARAPLEGIA OF ERB.**—Under this title Erb has described a class of cases with a clinical aspect sufficiently distinct to deserve special description, and of which syphilis is the invariable antecedent. It must be here stated, however, that Erb's type is not the common type of spastic paraplegia due to syphilis; acute myelitis with consecutive spastic paraplegia being a much more common result of luetic infection.

Males are much more frequently affected than females. The onset is insidious, and occurs in half the cases within three years of infection, and in almost all cases within five years. The symptoms are those of progressive spastic paraplegia, the upper limit of the affected regions corresponding with the lower half of the thoracic spinal cord. Dull aching pain in the back, often affecting also the legs, is an obtrusive and troublesome symptom. The most distinctive sign is great increase of the tendon reflexes, often the presence of epileptoid spasms and much rigidity, notwithstanding which fair voluntary power is preserved, and the patient may be able to walk with an ease which is surprising. Slight changes in common sensibility are present, and the sphincters are always affected, but usually not to a severe degree. These cases usually improve under treatment, and the symptoms may entirely disappear.

*Morbid Anatomy.*—The condition is believed to depend upon a local syphilitic infiltration of the pia arachnoid and superficial parts of the spinal cord, with small foci of thrombosis in the substance of the cord.

*Diagnosis.*—This type is distinguished by a history of antecedent syphilis; by the gradual onset, chronic pain, preservation of voluntary movement with spastic paraplegia, sphincter trouble, and a tendency to improve greatly under treatment.

*Treatment.*—Energetic antisyphilitic treatment with the most careful attention to the improvement of general nutrition is necessary. Mercury is indispensable and should be administered by inunction. Strychnine should be avoided. Continuous rest in bed should be avoided, as the power of walking soon becomes impaired from the lack of practice. When improvement sets in massage and passive movement to the legs are most beneficial, and Fraenckel's exercises are very useful.

**INFANTILE SPASTIC PARALYSIS.**—Spastic paralysis occurring in children is most frequently due to affections of the brain, less com-



monly to lesions of the spinal cord. In the former the lesion may be bilateral or unilateral, and the resulting paralysis, diplegia or hemiplegia. In the latter case the paralysis is segmental and paraplegic in distribution. The resulting groups, (1) Cerebral diplegia, (2) Infantile hemiplegia, and infantile paraplegia, are further separated in that each constituting a distinct pathological entity bears no pathological relation with the others.

**CEREBRAL DIPLEGIA.**—(*Syn.*) Little's disease; Congenital spastic paraplegia; Birth palsy.

The term cerebral diplegia comprehends a group of diseases for the most part congenitally installed, with a somewhat varied clinical aspect, but in all of which spastic paralysis, frequently associated with perverse movement, is distributed upon both sides of the body. Mental impairment and recurring convulsions are frequently associated. A widely spread, diffuse lesion of the cerebral cortex of both hemispheres is invariably present.

The clinical picture of this group was first described by Little in 1853, who was the first to show its association with premature birth. The use of the term "birth palsy" by subsequent writers most unfortunately led to the erroneous connection of this disease with cerebral injuries received during birth. The misinterpretation of an important pathological communication by MacNutt in 1888 led to the association of this disease in England with meningeal hæmorrhage. Further pathological investigation has led to the abandonment of meningeal hæmorrhage occurring during birth as a cause of cerebral diplegia, which has been proved to depend upon a primary degenerative process occurring in the cerebral cortex.

*Causation.*—The symptoms of the disease are in rare instances evident at birth. More frequently some abnormality of movement first calls the attention of the nurse or parents some weeks after birth. Again, when the time arrives for a child to walk or to talk, his bodily defect may for the first time become apparent. In all cases appearing before the third year it is probable that the disease is congenitally installed. After the third year the occurrence of diplegia is very rare. Such cases of acquired diplegia do however occur, and have been met with as late as the fourth decade of life. Among the subjects of this disease males preponderate slightly. Heredity as a rule plays no part in the causation of the disease, but instances of a mother afflicted with congenital diplegia giving birth to similarly affected children are recorded.

The known etiological factors of congenital diplegia are maternal pathological states. A history of ill-health of the mother during pregnancy is common in cases of diplegia. The mother often volunteers the statement that while she was perfectly well and strong during her other pregnancies, her health failed completely during the carriage of the child afflicted with diplegia. Overwork and privation sometimes account for this failure of health. Several recorded cases give a history that a woman after bearing healthy children suffers with symptoms which may be described as those of chronic neurasthenia. She then gives birth to a child afflicted with diplegia, and her subsequent pregnancies end in still-births. This seems to show a gradual failure in the physiological process of generation, and it has been pointed out by most authors who have written on the subject that the youngest members of large families are especially prone to suffer. Not uncommonly several children of the same mother are affected, and it has been repeatedly observed that where one child of a family is afflicted with



diplegia, the mortality among the other children is remarkable, and premature birth is common.

In some cases a pathological psychic state of the mother during the time of pregnancy has been noted. Acute diseases and acute specific fevers occurring during pregnancy are of great importance as etiological factors in this disease.

Syphilis is a most important and by far the most common maternal disease standing as a causal antecedent to cerebral diplegia.

Premature birth and precipitate labour; difficult labour and asphyxia neonatorum are frequent associated conditions, and until recently were held as the essential factors of the disease. It is, however, only an infinitesimal minority of prematurely born children who become afflicted with diplegia, and the difficulty of labour associated with diplegia is not that due to obstruction in the parturient path, but that due to uterine inertia, itself a sign of an abnormal maternal state. It is highly probable, therefore, that abnormalities of birth stand not in causal relation with the disease, but are concomitant effects of a morbid maternal state.

In the consideration of cases of cerebral diplegia, arising after birth, stress must be laid upon the fact that the post-natal appearance of symptoms of disease is no proof that the disease was not congenitally determined. The possibility of an acute disease immediately preceding the onset of symptoms being in some cases only an immediate exciting cause, bringing into evidence a latent pathological condition, must also be borne in mind.

Most cases of post-natal cerebral diplegia do not follow any known acute disease. Cases have been reported in which the onset of symptoms directly followed the occurrence of the following acute diseases: gastro-enteritis, scarlet-fever, measles, mumps, pneumonia, acute rheumatism, typhoid, diphtheria, and influenza.

The clinical picture of cerebral diplegia, in all its forms, presents a combination, in varying degrees, of certain characteristic symptoms always bilaterally represented, though often more severe upon one side of the body than the other. These symptoms are: muscular rigidity, paresis, perverse movements, contractures, and increased deep reflexes. Mental deficiency is commonly present.

According as one or more of these symptoms preponderates over the other and dominates the clinical picture, certain striking types may be distinguished:—(1) General rigidity, where the spasm and paresis are more or less uniformly distributed throughout the whole body. (2) Paraplegic rigidity, where the lower extremities alone are markedly affected. (3) Bilateral athetosis, where slow perverse movement, occurring chiefly in the peripheral part of the limbs, is obtrusive. (4) Choreiform diplegia (congenital chorea), where the perverse movement is quick and occurs chiefly at the proximal joints of the limbs. (5) Congenital spastic idiocy, where the mental deficiency is very marked, and the other symptoms slight. One-half of all cases of diplegia are of the type “general rigidity,” and one-quarter “paraplegic rigidity”;—the remaining fourth being distributed equally between the other types.

*Clinical Aspect.*—In the majority of cases of generalised rigidity and in athetotic and choreic diplegia, the symptoms date from birth. Often in slighter cases the symptoms are only manifested when the child begins to crawl about or to walk. The attention of the parents may be drawn to the fact that something is wrong with the child by its being backward in talking and mentally unlike other children, and examination may then reveal slight



generalised or paraplegic rigidity. The nurse, in washing the child, is often the first to notice the unnatural stiffness of the limbs. In congenital athetotic and choreic forms, the symptoms become much more evident when the child begins to get about. In cases where the symptoms are noticed first some time after birth, a careful attention to the history of the case will generally reveal some abnormality of the child, indicating that the case was congenital, and separating it from cases of post-natal origin. Such symptoms, as difficulty of swallowing, strabismus, nystagmus, slight perverse movements (choreic and athetotic), slight rigidity, the regular assumption of an unnatural attitude, and pes cavus, are likely to be noticed by those who tend the infant, and are of great value in that they may be the earliest signs of disease to attract attention.

In post-natal cases the disease is often ushered in by a group of symptoms resembling the prodromal symptoms of acute anterior poliomyelitis, namely, malaise, anorexia, headache, slight fever, and sometimes vomiting. These symptoms last from one to four days, after which the symptoms of diplegia gradually make their appearance. The onset of the disease is frequently with convulsions, either a single attack of convulsion or repeated attacks. After such convulsions the symptoms of diplegia may come on slowly, or may develop from a condition of general paresis of the limbs and trunk which immediately follows the convulsive attack. Sometimes both convulsions and febrile symptoms, lasting a few days, occur at the commencement of the disease, and this has been more often noticed in cases of post-natal paraplegic rigidity. In other cases the onset is gradual, and is not accompanied either by convulsions or febrile symptoms, and it may be many weeks before the symptoms reach moderate intensity. Such chronic onsets sometimes date from some trauma to the head, often of a trifling nature. A slow onset without definite initial symptoms is the rule in cases commencing after childhood.

The course of the disease, speaking generally of all types, may be either regularly progressive, or the symptoms, having reached a certain degree of intensity, may either remain stationary or tend to some slight amelioration. In severe cases of generalised rigidity, and in most athetotic forms, a progressive course is the rule, and very few patients so afflicted reach adult life.

A tendency to amelioration is sometimes seen in cases of slight generalised rigidity and in cases of paraplegic rigidity, but even in the slightest cases the amount of improvement is not great, and the symptoms never entirely disappear.

Between the several types of cerebral diplegia there exists every variety of transitional form and combination.

*Generalised Rigidity.*—As its name implies, the chief characteristic of this type of diplegia is a condition of muscular rigidity associated with weakness affecting to a greater or less extent all the muscles of the body. Except in the most severe cases, where the weakness amounts to complete paralysis, there is more rigidity than weakness. In some cases it is astonishing that there should be so much power in the presence of such a degree of rigidity. The distribution of the rigidity and weakness is generally that the lower extremities are most affected, the upper to a less degree, and the facial region still less. In a few cases the arms have been more affected than the legs. Movement is slow and clumsy, and often athetotic, and choreic movements are present in the limbs. If the patient is able to walk, the gait is digitigrade from contracture of the calf muscles. The knees are slightly flexed from contracture of the hamstrings. The



thighs are rotated inwards and the knees pressed close together, rubbing against one another with each projection of the limb. More severe adductor spasm gives rise commonly to the "cross-legged" progression.

The assumption of the sitting position is impossible in marked cases. When such a patient is placed in a chair the legs stand out stiffly, and the child will not fit the chair, owing to the rigidity preventing the necessary flexion at the junction of the lower extremities with the trunk; he tends, therefore, to slide off. In the most severe cases the patient lies in a log-like fashion, unable to move or turn in bed, and then the rigidity may reach such a degree that he may be lifted by the heels in one rigid piece, the weight of the body being supported on the back of the head. In this stage, owing to muscular contractures, certain characteristic attitudes are assumed which will be described later on.

*Mental Symptoms.*—Every degree of mental impairment is met with in these cases, from slight mental dulness to complete amentia. As a rule the average degree of mental impairment is more profound than in other types of diplegia. It, however, does not always correspond to the severity of the rigidity and paralysis. The most profound mental deficiency may exist in cases where the rigidity and paralysis are quite slight and *vice versa*. This corresponds with the greater or less affection of the præ-frontal lobes.

*Speech Defects.*—In congenital cases, and in cases occurring before the eighteenth month, speech may never be acquired, or may be much delayed, and when acquired is often imperfect. Articulation is imperfect, slowness of speech, with great facial over-action, slurring, and stammering often occur. When the disease arises after speech is learnt this is generally completely lost if the child be younger than six years of age, and if the case be at all severe. The loss of speech may be quite gradual, and where some intelligence persists, the understanding of words may remain in patients who can utter no articulate sound.

*Convulsions.*—With this type of cerebral diplegia convulsive attacks are more commonly associated than with the other types, choreic diplegia and bilateral spastic hemiplegia being excepted. They occur in rather more than half the cases.

*Cranial Nerves.*—Primary optic atrophy occurs in a considerable number of cases. It may produce complete blindness.

Inequality of the pupils and slowness of the light reaction are not uncommon. Spontaneous nystagmus of very wide range is common in cases where the face is markedly affected. It is to be regarded as the spastic ocular sign analogous to the rigidity elsewhere, and produced in the same manner.

Convergent strabismus occurs in about 30 per cent of the cases.

In nearly all cases the face shows signs of paresis and rigidity. With slight involvement of the face this may be shown by a general loss of emotional expression, slight retraction of the eyelids, and some retraction of the angles of the mouth. With more severe involvement of the face, an unvarying starchy expression, with wide palpebral apertures and a large open mouth (*bouche en cœur*) are present. Slobbering is common. In some cases involuntary facial overaction is present, and gives rise to varying grimaces on attempts to move the face or on speaking. Athetoid movements of the face are not rarely seen.

The tongue is often very large and the hard palate very much arched. The presence of adenoid vegetations in the pharynx is frequently noticed.

Difficulty of swallowing is present in the large majority of cases, though it may be slight. On watching the act of deglutition in these cases the



difficulty seems due to rigidity of the muscular apparatus rather than to any paralysis.

Laryngeal difficulties, dysphonia, stridor, etc., have been occasionally noticed.

*Conformity of the Skull.*—In congenital cases microcephaly is generally present (25 per cent of all cases). Signs of hydrocephalus are present in some cases. Asymmetry and flattening of the region of the central convolutions are common.

In a few cases a defect in the skull in the central region, through which the brain could be felt to pulsate, has been found, and autopsies upon such cases have shown a porencephalic defect underlying the hole in the skull.

*Attitude.*—In the more severe cases characteristic attitudes are assumed. The head may be rigidly retracted, but more commonly the chin is pressed down upon the chest. Torticollis has been noticed in two cases by Rosenthal.

The spinal column generally shows kyphosis, sometimes lordosis, and often scoliosis.

The cases presenting lordosis usually have head retraction, and those with kyphosis a flexed position of the head.

Several characteristic attitudes have been described and named, and these occur with sufficient frequency to deserve special note.

(1) *The Cramped Attitude.*—The head and trunk are bent forward. There is kyphosis. The elbows are pressed into the sides and the forearms crossed on the chest in contracture. There is contracture in the fully-flexed position at hip and knee joints. The position has been described as that in which the patient occupies the least possible space.

(2) *The Attitude of Adoration.*—The head is retracted and the eyes are staring. There is lordosis. The elbows are pressed into the sides, and the hands are held supinated below the face. The legs are rigidly extended.

(3) *The Extension-supination Position.*—The limbs are fully extended and the forearms are fully supinated. More rarely extreme pronation is associated with the extended position of the limbs.

Pes cavus is present in most of the cases. Some cases show a condition of talipes equino-varus. Genu recurvatum sometimes occurs.

The mode of progression in these cases, where any is possible, is characteristic. Little compared the gait to that of the tardigrade animals. The "cross-legged" progression is so well known as to need no description here. Where the legs are so severely affected as to render walking impossible, while fair power remains in the upper limbs, the patient may crawl along the ground with the upper limbs, dragging the useless lower limbs and trunk.

*Muscles.*—In some cases a curious hardness and resistant feeling of the affected muscles is present, resembling a similar condition in pseudo-hypertrophic paralysis. If athetosis is associated with the rigidity, there may be actual hypertrophy of the muscles.

In late stages of severe generalised rigidity the muscles are always wasted.

*Perverse Movements.*—Under this heading must be classed the very constant maladroitness of movement present in these cases, the facial overaction and grimacing in speech and in mimetic expression; choreic movements and athetotic movements and intention tremor. These forms of perverse movements are only present where the rigidity is absent. Athetotic movements sometimes are present in the face; choreic movements never. Not rarely choreic or athetotic movements are present in the upper

extremities, associated with slight rigidity, while severe rigidity affects the lower limbs.

Choreic movements may develop *pari passu* with the rigidity. A case of generalised rigidity may clear up for the most part, leaving a choreic diplegia. Athetotic movements always appear at a later date than rigidity. Cases presenting either choreic or athetotic movements are said never to improve after such movements have made their appearance.

Tremor, precisely resembling that seen in disseminated sclerosis, is sometimes seen in limbs where the rigidity is slight. Probably some of the so-called cases of disseminated sclerosis in children are in reality cases of diplegia.

*Common sensation* and the muscular sense are unimpaired in the cases where their accurate investigation is possible.

In the severe cases there is very great pain on passive movement of the rigid joints, probably owing to the formation of rest adhesions.

Trophic changes are confined to a stunting of growth, but this is not nearly so well marked in the diplegias as in infantile hemiplegia and in bilateral spastic hemiplegia. In diplegia the bilaterality of the distribution renders it difficult to estimate.

The sphincters are unaffected.

The deep reflexes are exaggerated, but are difficult to obtain in limbs which are very rigid, and for this reason foot clonus is not generally obtained. Of the superficial reflexes those of the trunk are absent in marked cases. The plantar reflexes are usually of the extensor type, but sometimes of the flexor type.

*Congenital Spastic Idiocy*.—These cases are placed under a special heading because of their peculiar clinical aspect. They present slight symptoms of generalised rigidity with profound mental changes, which generally amount to complete idiocy. In these cases the incidence of the cortical lesion is chiefly upon the frontal convolutions.

*Paraplegic Rigidity—Little's Disease*.—In this type the rigidity and paresis are confined to the lower extremities. Tardigrade progression, or the cross-legged progression, or the "broken-backed" progression, is present. Adductor and extensor spasm of the legs with pes cavus is the rule. Not rarely there is hyperextension of the knees. The general description given in the description of generalised rigidity applies, as regards the lower extremities, to this type.

Convergent strabismus occurs more frequently in this type than in the others, and is found in 30 per cent of the cases.

Mental defects, difficulties of speech, and of swallowing occur as frequently as in other types, but for the most part in a lesser degree of severity.

Convulsions are present in 36 per cent of the cases.

Combinations of paraplegic rigidity, with choreic diplegia and with bilateral athetosis, occur.

*Bilateral Athetosis and Choreic Diplegia (Congenital Chorea)*.—The essential feature of the types consists in the presence of constantly occurring spontaneous movements which may affect all the muscles of the body. Bilateral athetosis and choreic diplegia are only separated as types by the form and extent of the spontaneous movements, and the two forms of movement may occur in the same case.

The movements of athetosis are slow, rhythmical, and affect the peripheral parts of the limbs most.

The choreic movements are quick, shock-like, localised, and affect the proximal parts of the limbs most. The term "choreic movements" is an



unfortunate one, as the movements in no way resemble those of true chorea.

The trunk muscles are often affected. Choreic movements always cease during sleep.

Athetosis frequently affects the face, and is associated with great facial overaction and grimacing on voluntary movement. The tongue, when at rest in the mouth and when protruded, may show most evident athetosis. In severe cases all the muscles of the body may be affected, and the movements beginning in one limb may spread successively to the other limb on the same side, and then to the opposite limbs. In some cases the movements are confined to one region of the body. The face or the upper or lower extremities may be affected alone. Athetotic movements sometimes continue during sleep. Slight generalised rigidity and paresis are present in these cases, though they may amount to little more than an increase of the deep reflexes and slowness of voluntary movement. The same mental changes, affection of speech, nystagmus, strabismus, optic atrophy, and dysphagia, as have been described in "generalised rigidity," occur in bilateral athetosis and in choreic diplegia. A few points, however, demand special notice. Even in the most severe cases of bilateral athetosis the mental changes may be very slight. Convulsions are common in choreic diplegia, but are very rare in cases of bilateral athetosis. In both types intention tremor is not infrequently noticed. In athetosis actual hypertrophy of the muscles is sometimes seen.

*Course of the Disease.*—Some cases are regularly progressive, either rapidly or slowly, and this generally is the case with the more severe forms of generalised rigidity. Others, after the symptoms have reached a certain degree of intensity, may remain stationary, or may tend to improve. The latter result is more often seen in cases of paraplegic rigidity and slight generalised rigidity. The amount of improvement is, however, rarely great. In slight cases of generalised rigidity the symptoms may disappear from the upper limbs, leaving a condition of paraplegic rigidity.

Improvement is, perhaps, never met with in bilateral athetosis and choreic diplegia, and cases of athetosis are usually progressive.

Speaking generally, cerebral diplegia tends to shorten life much more than does hemiplegia, and few infantile cases reach adult life.

*Pathology.*—Though a considerable number of autopsies have been recorded, the examination has been made long after the onset of the disease, and from the condition found it has been impossible to infer the initial lesion. In nearly all the cases there has been symmetrical atrophy and sclerosis of the convolutions of both cerebral hemispheres, without disturbance of their form and arrangement. The atrophy always affects the Rolandic region to a greater or less extent. The convolutions are small and hard, and the sulci widened. The cortex is thinned, and its surface often presents a worm-eaten appearance. The surface of the brain generally presents an appearance like that of a walnut kernel. Often associated with this atrophy are cysts, sometimes multiple and small, sometimes large, a condition of porencephaly being present. There is usually some dilatation of the ventricles. Microscopically there is atrophy of the nerve cells of the gray matter, and in proportion as the case is of long standing or recent, there is more or less increase of the glial tissue and periarterial thickening.

Many different pathological processes have been held responsible for the atrophy of the cortex. Meningeal hæmorrhage occurring at birth, venous and arterial thrombosis in the cerebral cortex, embolism, and in cases where cysts exist, cerebral hæmorrhages, have been brought forward as the primary



lesions, but with increasing investigation the evidence has gone against these factors. Non-development of the pyramidal system is rather the result than the cause of the disease, as some authorities hold. Polio-encephalitis has been proved to produce diplegia only in adults, and the clinical picture of this rare disease is peculiar. There is much evidence that the condition depends upon a primary degeneration of the nerve cells of the cerebral cortex, and it is probable that this degeneration is caused by some as yet unknown toxic agent.

*Differential Diagnosis.*—The diseases most likely to be confounded with cerebral diplegia are Marie's hereditary ataxy, Friedreich's ataxy, disseminate sclerosis occurring in children, and familial spastic paralysis.

*Treatment.*—Only the slighter forms of generalised and paraplegic rigidity afford any scope for treatment.

Careful mental training and the regular use of appropriate gymnastic exercises may do much to aid improvement.

Slow extension and the division of tendons in slight cases where contractures have occurred have produced encouraging results.

*Infantile Cerebral Degeneration.*—Nearly allied to the disease just described are a remarkable and very characteristic group of cases, first described by Warren Tay, to which the above name has been applied.<sup>4</sup>

In the reported cases of this disease the patients have all been Hebrews. The disease is familial. There have been no clinical antecedents. Birth has always been natural and at full term. The patients have been healthy till between the third and sixth months, when general weakness comes on insidiously, and is followed some time later, if the child live, by general muscular wasting and rigidity. The limbs may ultimately become quite flaccid. Great mental failure, optic atrophy, and peculiar retinal changes have been present. The retinal changes are in the macular region, where a whitish gray oval patch is seen about twice the size of the optic disc, with its long axis lying horizontally, and its surface slightly raised above the general surface. The fovea centralis is seen as a cherry red spot in the centre of this area. The disease has proved fatal in all cases at an early period, generally before the second year of life.

A progressive primary degeneration of the large cells of the cerebral cortex has been constantly found in these cases. A similar degeneration of the cells of the retina has been also present.

*INFANTILE HEMIPLEGIA.*—While according to the strict meaning of the term "infantile" this group should comprise all cases of hemiplegia occurring before the age of puberty, it has become broadly restricted to cases occurring before the sixth year of life. There are several reasons for this limitation. Either from the peculiarity of the pathological process underlying the disease, or from some property of the cerebral tissues of children to retain in part their vitality after injury, the post-mortem appearances of the brain in cases of infantile hemiplegia are strikingly different from those met with in adult hemiplegia. Again, local injury occurring during the period of active growth and before the taking up of function is complete, is more easily compensated by the taking up of a function by the uninjured parts. It must be pointed out, however, that while from such compensation functions of the infantile brain are better preserved in the destruction of limited areas by disease than are those of the adult brain, widespread lesions produce far more loss of function than occurs in the adult brain, great mental impairment resulting.

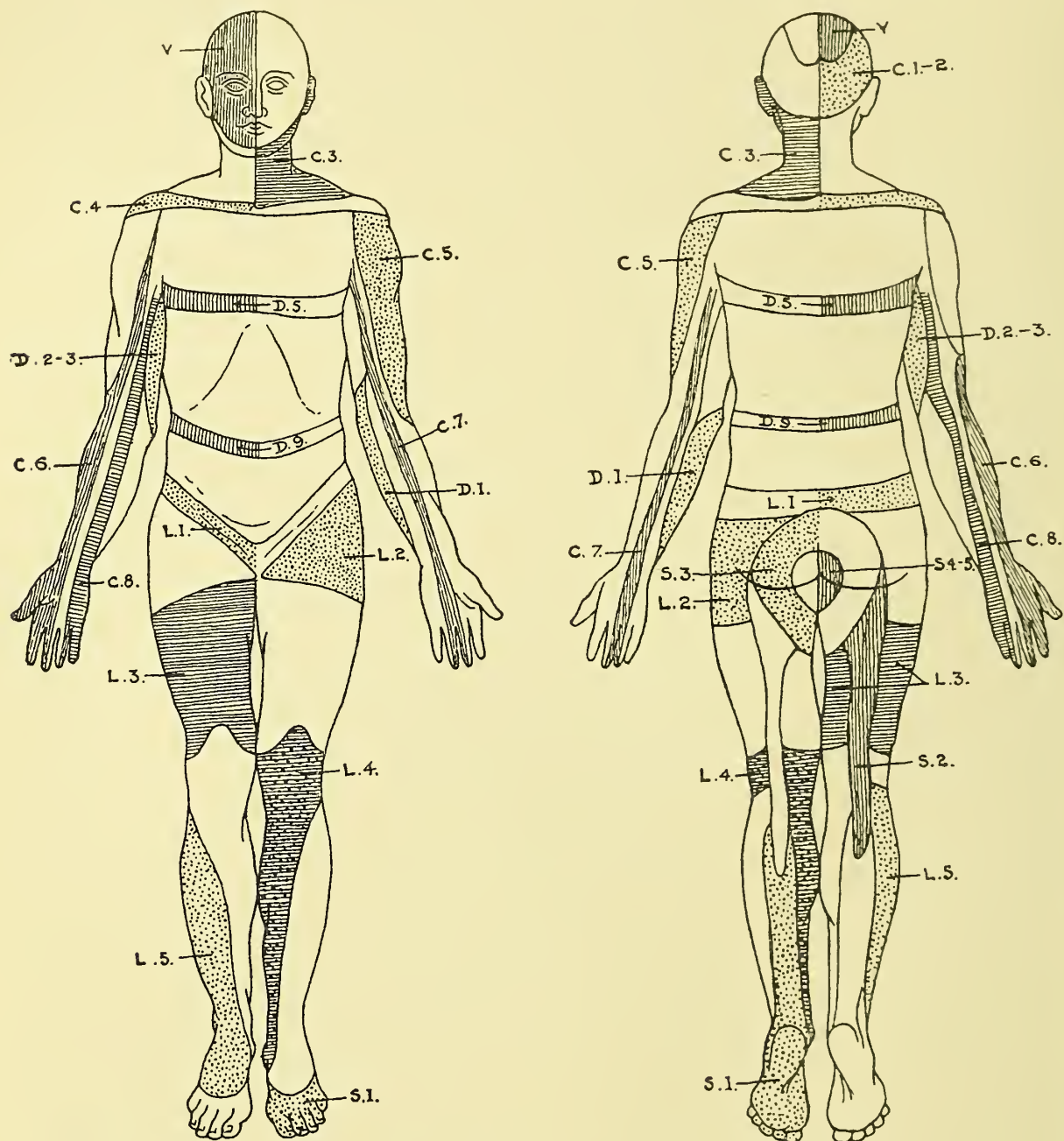
Further, clinically, infantile hemiplegia is characterised by stunting of



growth and often by athetosis on the paralysed side; by the absence of aphasia, and by the occurrence of recurring convulsions in many cases.

*Causation.*—In a few cases infantile hemiplegia is of intra-uterine origin, and some cases dating from birth are due to cerebral injuries received during parturition where labour is obstructed, and where instrumental delivery is necessary.

In the majority of the cases—75 per cent—the onset is during the first



(After Collier and Purves Stewart.)

three years of life, and in 60 per cent during the first two years. Females preponderate slightly over males.

Neuropathic heredity and parental syphilis do not appear to be in causal relation with the disease.

Most frequently the disease is not distinctly secondary to any known morbid influence. Less often it follows some acute disease, occurring either during the decline of the disease or within a fortnight of its termination. Scarlet fever, measles, and diarrhoea are the most common diseases associated with infantile hemiplegia. Other diseases are broncho-pneumonia, diphtheria, typhoid fever, and mumps. It sometimes occurs during a paroxysm in whooping-cough, presumably from cerebral hæmorrhage. It is almost unknown in connection with acute or chronic rheumatism.

*Clinical Aspect.*—The onset is always acute, and is attended in more than half the cases with severe convulsion and loss of consciousness, which may last for days. The hemiplegia may be at once apparent when the child regains consciousness, or it may not show itself until several attacks of convulsions, separated perhaps by days, have occurred. It may then increase in severity with each attack until it becomes severe. Where initial convulsions are absent the child is seized with fever, vomits, and becomes prostrate or comatose, and on recovering, the hemiplegia is first noticed. In older children headache is usually complained of, and transient aphasia may occur if the hemiplegia be on the right side, but permanent aphasia does not occur in children under six years of age unless a very severe degree of mental impairment is associated.

The hemiplegia, which is more common upon the right side, may be complete at first, and in children who can speak hemianæsthesia may sometimes be demonstrated, but it is rapidly transient. Soon the paralysis lessens, and the persistent condition, after the lapse of time, varies in different cases from an incapacitating degree of hemiplegia to complete recovery. Very rarely the hemiplegia is bilateral; it is then profound, and little improvement takes place. The ultimate condition depends upon the severity of the symptoms first manifest. In most cases the face shows little permanent paralysis, but there is often some overaction and involuntary grimacing upon the affected side. Both the face and the tongue may show athetosis. Not infrequently there is convergent strabismus. The arm is always more affected than the leg, and power is recovered most at the shoulder and least in the fingers. Post-hemiplegic perverse movements are frequently met with in the arm, rarely in the leg. Slow, spontaneous rhythmic movements (athetosis) occur chiefly at the peripheral joints of the limb, and are seen more typically in this disease than in any other condition. They are most marked when they occur in a completely paralysed limb. When some volitional movement remains, athetosis may sometimes be controlled voluntarily. Short, sharp involuntary movements are of rare occurrence; they occur in limbs where considerable voluntary power has been regained, and affect chiefly the proximal parts of the limb (shoulder and hip). Such movements have been called post-hemiplegic chorea. As perverse movements also must be classed the slowness and clumsiness with which almost all the movements of the hemiplegic side are carried out.

Where any considerable degree of hemiplegia persists the paralysed muscles soon become hypertonic, and rigidity sets in with contractions at the joints, and the assumption of peculiar positions of the limbs. In the upper limb the elbow is usually flexed; the forearm pronated; the wrist flexed; the fingers flexed at the metacarpo-phalangeal joints and extended at the other joints, and the thumb adducted—contracture being present at all these joints.

In the lower limb the most consistent deformity is dropping of the foot and pes cavus, the Z-shaped outline of the foot seen from the inner side being characteristic. There is sometimes inversion of the foot. Some degree of kyphosis and scoliosis, with the concavity to the hemiplegic side in the dorso-lumbar region, is common. The pelvis is tilted down on the paralysed side if the patient is able to walk. The gait is usually that characteristic of hemiplegia, but it may be much modified by tricks which the child acquires for the purpose of overcoming his difficulty. The greatest attention must be paid to the peculiarities of voluntary movement and to the contractures, since it is by the persistent treatment of these by



training and surgical procedures that admirable results may be obtained in patients who when untreated never improve.

There is almost invariably marked stunting of growth on the affected side, involving the half of the body up to the middle line. Such stunting is present even in cases where the paralysis has almost entirely disappeared. There is most stunting of growth in the most paralysed parts. The difference between the two sides is often very obvious when the scapulæ and the two sides of the pelvis are compared. The shortness of the structures on one side of the body, especially of the lower limb, are responsible in part for the scoliosis, tilting of the pelvis, and dropping of the foot. When athetosis is present, however, the muscles involved in the spontaneous movement may be actually hypertrophied. The cranium, when asymmetrical, is usually smaller on the side corresponding to the cerebral lesion, and such deformity is accounted for by the non-occurrence of growth in the diseased cerebral hemisphere. Rarely the bone may be absent over the seat of the lesion, and a rounded aperture, through which the cerebral pulsation may be felt, may be present.

According as the damage to the brain is widely spread or not, and the prefrontal lobes more or less affected, infantile hemiplegia may be associated with every degree of mental impairment from idiocy to the slightest mental impairment. Nearly all cases show some intellectual deficiency on reaching puberty, but whether this is always due to the cerebral lesion, or sometimes to the lack of training that infirmity and backwardness entail, is not clear, for it is certain that by careful training in childhood it may be reduced in some cases to an unimportant amount. Mental failure is generally severe in cases where recurrent convulsions become established epilepsy.

Recurring convulsions are much more common in infantile than in adult hemiplegia.

Such convulsions usually date from the occurrence of the hemiplegia, but they may not occur till months or years after.

The convulsions may be general; more frequently they are confined to the affected side, and are then more common in left than in right hemiplegia. The onset is generally deliberate, often strictly local in its commencement, and consciousness is lost late. There may be transient increase of the paralysis after each attack.

Affections of the special senses, hemianopsia and hemianæsthesia, do not occur as lasting symptoms. Internal strabismus is not rare, otherwise the cranial nerves are unaffected.

The sphincters are not affected. Slight trophic changes in the skin of the peripheral parts of the affected limbs are sometimes present, and in some degree vaso-motor paralysis is common.

The superficial reflexes are generally diminished upon the hemiplegic side, and the plantar reflexes on that side are of the extensor type; this extensor reflex is always marked where a pes cavus is present, but sometimes when there is a dropped foot, with toes rigid in the flexed position, a flexor type of reflex obtains. The tendon reflexes are exaggerated upon the affected side, and foot clonus is obtainable. Rigidity may, however, cause these signs to be difficult of elicitation. The knee-jerk upon the sound side is often more brisk than normal.

*Prognosis.*—In the rarest event only is infantile hemiplegia fatal immediately after its onset, otherwise it has no tendency to destroy life nor to shorten life, except that the subjects of the malady offer less resistance, perhaps, to intercurrent diseases than do healthy children. Many cases improve rapidly, and the paralysis may leave slight clumsiness in moving



the arm or leg, or recurring unilateral convulsions, as the only sign of its previous existence. Where lasting paralysis, rigidity, and contracture exist, slow improvement is still the rule, and it is difficult to say when such improvement is likely to cease, as cases go on improving long after puberty, especially if treatment be properly carried out.

Mental impairment renders the prognosis of amelioration hopeless in proportion to its severity, as the re-education of movement upon which recovery greatly depends becomes impossible.

Cases where athetosis exists never regain useful movement in the part where the spontaneous movement occurs. Athetosis involves total permanent disablement in the region of its distribution. Recurring convulsions render the prognosis worse in proportion to their frequency, persistency, and tendency to temporarily increase the paralysis; further, such attacks are not infrequently associated with progressive mental deterioration. The probability of improvement is greater at a period close to the onset than at one more remote. If there be even the slightest voluntary power in all parts of a limb, however clumsy the movement, the probability that a useful working limb will result is great if treatment can be adequately carried out. If with a fair degree of intelligence present a child with infantile hemiplegia can walk at all, it may be safely said that he will walk well ultimately if treatment be properly carried out. Much, however, depends upon the treatment, which, as it is difficult, laborious, and of long duration, is too often abandoned or even unattempted, and the few years following the onset of the disease being neglected, the lapse of time places an impassable barrier before the efforts of the physician. Contractions and deformities do not in themselves influence prognosis.

Cases presenting slight mental impairment may improve considerably in this respect. The amount of impairment can only be judged by the progress made under the influence of careful education of a special kind.

*Pathology.*—Since infantile hemiplegia has little tendency to be fatal at an early period pathological examination has been confined almost entirely to periods long after the onset of the disease, when, from the occurrence of some secondary process (sclerosis, cyst formation, etc.), it is difficult to correctly surmise the nature of the initial morbid process. The condition found at these is shrinking and atrophy of the convolutions of a limited area of the brain. These convolutions are small and hard to finger, the sulci between them are widened, the sclerosed area being sunk below the level of the general surface of the hemisphere. After the removal of the membranes the surface of the atrophied area presents an appearance which may be likened to that of a walnut kernel. There is always compensatory increase of the subarachnoid fluid over this area, and frequently a compensatory dilatation of the lateral ventricle beneath. Often associated with this atrophic lobar sclerosis is cystic formation, and the term porencephaly has been applied to the condition. There are several varieties of the cystic condition. There may be a local complete absence of the wall of the ventricle, the subarachnoid space being continuous with the cavity of the ventricle, or the ventricle wall may consist only of the lining membrane of the ventricle and the pia-arachnoid, or lastly, a smooth-walled cyst may separate the ependyma from the pia-arachnoid. In some cases a single cyst in the substance of the hemisphere is found, with walls containing the products of the degeneration of blood pigment, and suggesting that the origin of such a cavity has been a hæmorrhage.

In the congenital cases a general hemiatrophy of the brain, with little sclerosis, is present.



In atrophic lobar sclerosis the microscopic appearances are disappearance of the nerve elements and increase of the neuroglial elements with the presence of many spider cells. The vessels present no conspicuous change.

The initial morbid processes which lead to these results is not certain. It must be remembered that the infantile brain does not react in the same way to injury as does the adult brain. A lesion which in the adult would produce a softening, produces atrophy and sclerosis in the child. Except in the congenital cases (hemiatrophy of brain) where developmental arrest is caused by some as yet unknown lesion, and in cases dating from birth injury, where there is local traumatic destruction of cerebral tissue, it seems probable that an initial vascular lesion is present. Thrombosis of superficial veins of the cerebrum, thrombosis of the superior longitudinal sinus, multiple thrombosis of small arteries, have undoubtedly been the initial lesions in isolated cases.

Polioencephalitis, embolism of the middle cerebral artery, and cerebral hæmorrhage have been found present, but only in cases where the clinical aspect has been somewhat different to that usual in infantile hemiplegia. A vascular thrombosis, therefore, appears to be the most probable initial lesion, but we are entirely ignorant of the pathological process which is responsible for such a lesion. It is probable, however, that this is peculiar and entirely distinct from the processes underlying thrombosis in the adult.

*Diagnosis.*—The disease may be distinguished from the other chief cause of spastic paralysis of hemiplegic distribution—intracranial tumour—by its rapid onset and slow amelioration, and by the absence of headache, vomiting, and optic neuritis. The not uncommon absence of this last sign in glioma situated in the pons Varolii must be borne in mind. The fact that the paralysis is strictly unilateral separates this disease from cerebral diplegia, and there is neither optic atrophy nor a familial tendency to the disease in infantile hemiplegia. The rapid muscular wasting, loss of the deep reflexes, and absence of Faradic excitability in the muscles, easily distinguish the rare cases of anterior poliomyelitis where the distribution is unilateral, affecting both arm and leg, and the paralysis severe.

*Treatment.*—Since in the early stage of the disease its nature is only manifest when improvement begins and the hemiplegia becomes manifest, the treatment at this period is that of acute illness in general—rest and careful nursing. The subsequent treatment which should be commenced as early as the physical state will allow, should at first be massage to the paralysed limbs. Electrical treatment is to be avoided, as children are as a rule so frightened by its application that the advantage gained from it is more than counterbalanced. Directly some power is regained, every attention should be paid to the re-education of the returning movement by physical exercise of the faulty limbs. In order to direct volition to the paralysed side it is sometimes advantageous to confine the upper limb of the sound side. Such physical exercises are almost impossible where there is much mental backwardness and lack of attention, and these deficiencies must be as far as possible removed by special education. Where considerable improvement has been attained, if the child has reached a suitable age, exercises in front of a mirror are often useful, for he is able to see for himself errors of position and movement, and strives to correct them. Where treatment has been neglected and contractures have arisen, excellent results are generally obtained by tenotomy, followed shortly by passive movements, and subsequently by exercises. From the way in which the deformities arise, it is at once clear that the prolonged use of rigid appliances for their correction is useless.



The treatment, in all but the slightest cases, must be prolonged over years, often many years, and its application requires the greatest patience and devotion, but except in those cases where mental impairment remains severe, the ultimate result is excellent. Too often, however, from neglect or from inability to procure the necessary means of treatment, the patient with infantile hemiplegia remains hopelessly crippled. After adult life is reached little further improvement can be expected.

**INFANTILE SPASTIC PARAPLEGIA OF SPINAL ORIGIN.**—Spastic paraplegia in children, due to a lesion of the spinal cord, is most frequently due to compression of the cord as the result of spinal caries. Apart from this cause the condition is somewhat rare. It is occasionally produced by the pressure of neoplasms arising in the bony structures of the vertebral column, or in the meninges, and it sometimes results from spina bifida.

Injuries to the spinal column during birth give rise to paraplegia, and to this group of cases the term “obstetrical paralysis” has been given. Lastly, it may be a result of acute myelitis. The symptomatology does not differ from that of spastic paraplegia in adults, except that distortion and deformities of the limbs and trunk are more marked, since the spasm acts during the period of most active growth and upon less resistant structures.

**Diagnosis.**—The distinction of spastic paraplegia of spinal origin from the paraplegic form of cerebral diplegia is most important and often somewhat difficult. The history of the appearance of the symptoms, their non-progressive character, the preponderance of rigidity over loss of power, and the presence of mental impairment, recurring convulsions, and strabismus are the signs by which a cerebral lesion should be recognised. The presence of local spinal symptoms, pain, tenderness, deformity, or of the signs of spina bifida; the presence of anæsthesia and sphincter trouble, and a progressive character of the symptoms, should distinguish paraplegia of spinal origin. In paraplegia due to *spinal caries* the distinctive signs of the latter condition may be almost completely absent when the disease affects the upper dorsal spine, for as this is the least mobile part of the vertebral column, rigidity in this situation is not easily detectable. Deformity may be absent, and the pain caused by movement and jarring of the spine so slight as to seem negligible. A further difficulty in diagnosis arises as the symptoms may for a long time be confined to motor structures and complete spastic paraplegia, without a trace of anæsthesia or of sphincter trouble, may persist for weeks without the latter symptoms becoming manifest. In these cases, however, careful observation over a considerable length of time will generally reveal the nature of a condition present.

Spinal tumours for the most part present little difficulty in diagnosis. The majority of them being malignant and of rapid growth, are characterised by severe pains, radiating along the distribution of the nerve roots of the involved area, with a subsequent rapid and progressive onset of spastic paraplegia, later anæsthesia, and at last, when the pressure is sufficient to entirely abrogate the physiological conductivity of the cord, the paraplegia becomes a flaccid one. Such tumours are most frequently sarcomata, rarely myxomata or lipomata.

In the so-called “obstetrical paralysis” there is almost always the history of breech delivery, with difficulty in the passage of the shoulders or the after coming head, to overcome which vigorous traction upon the trunk has been used. This may produce either fractured dislocation of the cervical or upper thoracic vertebræ, or subluxation with hæmorrhage into or around the spinal cord. Frequently the diagnosis is aided by the presence of atrophic



palsy, limited to the distribution of some nerve roots of the brachial plexus on one or both sides, the anterior roots being injured by the traction upon the vertebral column. In such injuries the lesion of the spinal cord is often complete, so that a complete flaccid paralysis results. Deformity of the vertebral column is not often present, for the fractures may become reduced after the damage to the spinal cord has occurred. If the spinal cord be injured above the fourth cervical segment, death ensues almost instantaneously from paralysis of the respiratory muscles.

The diagnosis of paraplegia, due to spina bifida, only presents difficulty when the spina bifida is occult. The weakness of the lower extremities here dates from the time when walking is learnt, and is as a rule slight. Peculiar deformities of the feet and trophic changes may be met with. A careful examination of the vertebral column, which will reveal absence of the spines and perhaps of the laminae at some spot, a tuft of hair or naevus in the mid-line of the lumbar region, may at once suggest the diagnosis. Acute myelitis is a rare condition in children, and occurs usually from the age of six years to puberty. Congenital syphilis may be present. The onset and clinical aspect resemble the same disease in adults, and the diagnosis presents no difficulty.

*Treatment.*—See Spinal caries, Spinal tumour, Spina bifida, Acute myelitis, etc.

**SUBACUTE COMBINED DEGENERATION OF THE CORD.**—*Definition.*—Under this title a disease of the spinal cord has been described in which tracts of different function are concomitantly affected, and in which a definite train of symptoms occur which render the diagnosis of the disease during life a comparatively easy matter.

The word subacute should distinguish this affection from others in which tracts of different function are concomitantly affected, since they are all more chronic in their course.

*Etiology.*—The age at which the disease most commonly occurs is during the fourth and fifth decades of life, although not a few cases have been reported both in older and younger people.

Women appear rather more liable to the affection than men, although the difference of incidence in the two sexes is not marked.

Heredity seems to play no part in the etiology of the disease, and as a rule the patients have been in good health until its onset.

The affection has followed on acute infections, such as influenza, and it has also occurred after prolonged diarrhoea and long-continued suppuration.

Syphilis can almost be excluded as an etiological factor, although it has been present in a few cases, and the same may be said of alcohol.

Anæmia has been supposed to cause the disease, but on this question very different views have been expressed. There is no doubt that anæmia is one of the most common features late in the course of the affection, but it is by no means always present during the earlier stages, although in some cases it has been known to precede the onset of spinal symptoms by several years. The ascertained facts make it probable that the spinal cord changes and the anæmia are due to a common cause, rather than that the spinal cord changes are due to the anæmia. The blood does not, in the majority of cases, show the changes which occur in pernicious anæmia, although in certain cases (estimated by Dana at 10 per cent) such a condition is undoubtedly present.

*Onset and Course.*—The onset of the disease is usually slow and insidious, but occasionally the symptoms may come on rapidly and may be ushered in by headache, vomiting, and pyrexia.



The disease may with advantage be described as having three stages, but it must not be supposed that all cases conform in every detail to the following description:—The first stage is that of slight spastic paraplegia, with ataxia and marked subjective sensations in the lower limbs. The second stage is that of severe spastic paraplegia, with marked anæsthesia in the legs and trunk; and the third stage is one of complete flaccid paralysis of the lower limbs, with loss of all forms of sensation up to a varying level on the trunk.

The duration of the above stages is subject to considerable variation; the first is usually long, lasting some fifteen months or more, but, exceptionally, it may be of quite short duration, viz. less than two months. The second also varies greatly in duration; in some it lasts only a month, and in others it is prolonged over many months. The third is, as a rule, short, lasting only a few weeks, although in a few cases recorded it has lasted many months. It will be obvious from the above statement that no exact time limit can be fixed for the disease, although it usually runs its course in about two years.

*Symptoms.*—During the first stage of the affection the earliest manifestations are subjective sensations of numbness and tingling in the toes and in the extremities of the fingers; following on these sensory symptoms slight spasticity and ataxia develop, the legs feel stiff and cold, and there is some dragging of the toes. Girdle sensation and lightning pains may be complained of, and there may be considerable difficulty in walking in the dark and in standing with the eyes closed. There may be a little pain in passing water, but, as a rule, at this stage there is no incontinence of urine. On examination of the patient no anæsthesia, analgesia, or hyperæsthesia can be determined; some loss of the sense of position may, however, be present, the gait is slightly spastic, and there is a tendency to stand with the legs wide apart so as to secure a wide base, and when the feet are placed close together and the eyes closed marked unsteadiness results. The act of attempting to pick up a pin is performed in a clumsy manner, and on attempting to touch the tip of the nose with the first finger, the eyes being closed, lack of co-ordination and intention tremor become evident. There is slight rigidity of the lower extremities, the knee-jerks are increased, ankle clonus may be present, and the plantar reflex gives the extensor response (Babinski's sign).

The superficial reflexes are present and the myotatic irritability of the muscles is increased. The pupils react well to light and on accommodation.

The transition from the first to the second stage of the disease is often rapid, and is marked by the patient losing the power to stand in the course of a few hours or days; the inability being due rather to the want of co-ordination in the muscles of the legs than to actual loss of power, for while lying in bed the patient is often able to move the legs with considerable power.

Definite anæsthesia now becomes marked, commencing at first over the feet and lower portions of the legs, and gradually extending up the limbs, while sensation in the tips of the fingers also becomes affected. At this stage the impairment of sensation does not follow a segmental distribution, but is rather of a peripheral type, the longest neurons being affected both in the arms and legs. Sensation to all forms of impressions may now be gradually lost to a variable height on the trunk, and the distribution of the anæsthesia becomes segmental in character. A girdle sensation in the mid-dorsal region is frequently complained of, and there may be severe inter-



costal pain, which may in rare cases be accompanied by herpes zoster and even by subcutaneous hæmorrhages in the distribution of a nerve root.

During this stage the nutrition of the patient remains fairly good, the muscles do not waste to a marked degree, they retain their myotatic irritability, and they react normally to faradism and galvanism. Irregular rises of temperature frequently occur, and the patients are liable to attacks of diarrhœa. The knee-jerks are exaggerated, ankle clonus is present, the plantars give an extensor response, and all the superficial reflexes remain active in spite of the anæsthesia and analgesia. The sphincters are often unaffected. Exceptional symptoms that occur during this stage are weakness of one or other muscle of the eye, slight nystagmus, and retinal hæmorrhages; while unilateral paresis of one vocal cord has once been recorded.

The mode of onset of the third stage is one of the most striking features of this disease, the spasticity formerly present is rapidly replaced by flaccidity, a change that may occur in a few hours or a few days.

The temperature always rises at the commencement of this stage, but it subsides when the flaccidity becomes fully established. The inability to perceive all forms of sensory impressions over the lower limbs and trunk now becomes absolute, and there is complete incontinence of urine and fæces. The knee-jerks are rapidly abolished, ankle clonus and patellar clonus disappear. The plantar reflex, however, still maintains its extensor character, and this, in association with the absent knee-jerk and flaccid paralysis, constitutes one of the most diagnostic features of the disease. The other superficial reflexes persist and are active. Œdema of the legs and trunk often occurs at this period. The muscles now rapidly waste, and if this stage is prolonged the wasting becomes extreme in the lower extremities, and the upper extremities become similarly affected at a later period.

The muscles rapidly lose their faradic excitability, and the galvanic excitability is reduced, but there is no polar change such as is seen in complete reaction of degeneration.

Delirium at night is common during the third stage, and the patient often has varied delusions, while convulsive attacks, which give rise to fine clonic movements, but without loss of consciousness, have been observed in some cases. The patient's condition at this stage is one of extreme asthenia.

Cystitis frequently occurs, and bedsores have formed in nearly every case. Paralysis of the abdominal and lower intercostal muscles occurs during this stage, and the diaphragm becomes weak, and finally ceases to act. Death may then result from failure of respiration, or there may be sudden syncope. It may, however, happen that the patient lingers on in a condition of profound anæmia and sepsis, and dies of asthenia.

Although the course of this disease, as above depicted, is in the main correct, yet there are some cases which do not follow such a definite course, and the lower extremities do not pass into the final flaccid stage, but remain rigid in a position of extreme flexion.

Another class of case, again, is that in which the nervous symptoms manifest themselves in the course of severe anæmia which may have persisted with remissions for three to four years. In these cases it not infrequently happens that the final flaccid stage is never reached, the patients dying while the limbs are still spastic, and the impairment of sensations may be still limited to the periphery of the limbs, or sometimes loss of the sense of position is the only sensory defect present.

The condition of the blood from these cases varies considerably; in



many of them it has the characters of a secondary anæmia; in other cases, however, the blood shows the characters which are usually associated with pernicious anæmia, for there are microcytes, macrocytes, nucleated, and vacuolated red cells, and the percentage of hæmoglobin is relatively high.

*Morbid Anatomy.*—The condition of the body at the time of death depends upon the length of the third stage of the disease, for when this has been prolonged the body is usually extremely emaciated; when, however, death has occurred earlier the body is often fat.

No naked eye changes are visible in the brain or external surface of the spinal cord, nor in the membranes, but on section of the spinal cord the gray areas of degeneration in the posterior and antero-lateral tracts are plainly visible. The liver, which is usually enlarged, sometimes gives the free iron reaction, the kidneys not infrequently show a condition of septic infarcts or pyonephrosis secondary to the infection of the bladder, and the condition of the bone marrow varies considerably, in some cases being red and almost diffuent, while in others it is normal in appearance. The morbid changes in the spinal cord are among the most constant features of the disease, varying only in degree with its duration.

The stress of the disease falls upon the mid-dorsal region of the cord, and on examining sections from this level marked destruction of the white matter is seen all round the periphery of the cord, while the gray matter and a small area of white matter immediately surrounding it remain practically normal. If from this point in the mid-dorsal region the process is traced up the cord, it is found that the general destruction becomes gradually less, and instead of affecting all tracts alike tends to limit itself to the posterior columns, the dorsal and ventral cerebellar tracts, and the crossed pyramidal tracts. With regard to the affection of the posterior columns, the postero-internal (Goll) are much more affected than the postero-external (Burdach). But even in the cervical region, although the tracts above-mentioned may be the most affected areas of the cord, yet the disease is not limited to them, for scattered areas of degeneration are present, a favourite situation for these being in the ventral region of the cord in the position occupied by the direct pyramidal tracts and the neighbouring ground bundles.

The degeneration of the posterior columns can be traced up to the posterior column nuclei, and that in the dorsal and ventral cerebellar tracts into the inferior cerebellar peduncle and velum respectively, although with regard to the latter tract the degeneration can only be traced by the Marchi method—a method which reveals nerve fibres that are in a recent state of degeneration.

The degeneration in the pyramidal tracts can only as a rule be traced as high as the middle of the pons, although in exceptional cases degenerate fibres can be found also in the internal capsule and corona radiata. Sections from the lumbar region of the cord show sclerosis of the posterior columns and of the crossed pyramidal tracts, while in the sacral region the degeneration is almost limited to the crossed pyramidal tracts; but although this is in the main true, even in this region, scattered areas of degeneration are present outside the limits of these tracts. The walls of the blood-vessels in the sclerotic areas are thickened, but little change can be seen in them outside the affected areas. Very little change can be found in the gray matter or in the cells of the anterior horns, in those of Clarke's column, or in those of the cerebral cortex. The anterior and posterior roots appear normal, as do the posterior root ganglia, with the exception of rare instances



when changes have been found in them in connection with the occurrence of herpes or subcutaneous hæmorrhages. In cases that have reached an advanced stage considerable degeneration can be found in the nerves and marked atrophy of the muscle fibres, although in cases that come to autopsy earlier neither of these changes are present.

Two distinct processes account for the changes found in the spinal cord: a focal destructive lesion and a system lesion.

With regard to the first of these, an examination of the margin of the areas of degeneration reveals that the earliest change is swelling of the medullated sheath; within this outer zone the sheath can be seen undergoing fatty degeneration; this in turn becomes absorbed, and at the same time the axis cylinders disappear and a space is thus formed. By the grouping and fusion of these spaces vacuolated areas are formed which are very characteristic of the focal destructive process.

With regard to the second, viz. the system degeneration, the long tracts of the cord exhibit a degeneration similar to that found after a transverse lesion, and the tracts thus affected show a very definite sclerosis. The two processes merge very closely into one another, so that it is often difficult to say which is due to the focal lesion and which to the system lesion. It must not, however, be supposed that the degeneration is limited to the long afferent or efferent tracts of the cord, for it also affects fibres outside the region of these tracts.

*Pathology.*—Various views have been advanced as to the pathology of this affection: it has been thought by some that the cord changes were secondary to the anæmia; by others, that they were due to the occurrence of multiple hæmorrhages; by others, again, to changes in the vessel walls. The view, however, which seems to be most in accordance with the symptoms and pathological anatomy is that which attributes the condition to a toxic agent, which is jointly responsible for the changes in the spinal cord and for the anæmia. That the condition is, in part at all events, dependent on the vascular supply is shown by the fact that the degeneration is for the most part in the area of the cord supplied by the pial vessels. This, however, cannot be the only factor, and there is no doubt that the longer neurons tend to suffer more than those which are shorter. Neither of the above points, however, explain why the dorsal part of the cord is more liable to suffer than the cervical or lumbar regions.

*Diagnosis.*—The disease is recognised by the character of its onset, the association of ataxia and spasticity, and its progressive course. The differential diagnosis, although a simple matter in the later stages, presents considerable difficulties in the earlier stages of the affection. The two diseases with which it is most liable to be confused are tabes and disseminated sclerosis, and it will be well to deal with these first, and to afterwards consider the other affections which may possibly give rise to errors in diagnosis.

The symptoms during the earlier stages of the affection which suggest a diagnosis of tabes are ataxia and girdle pains, while in the later stages the loss of knee-jerk with flaccid paralysis of the lower limbs are those which bear the most striking resemblance to that disease. During the first stage the diagnosis depends upon the spastic condition of the legs, the exaggerated knee-jerks, the presence of ankle clonus, and the absence of the Argyll-Robertson pupil phenomenon. In the later stages when the knee-jerks are absent, the complete loss of power of movement in the legs, the absence of the pupil phenomenon, and the presence of an extensor response as the plantar reflex, together with the history of a previous spastic stage, make the diagnosis clear.



The symptoms that most closely resemble those of disseminated sclerosis are the slight nystagmus, ataxia, and spasticity, and the differential diagnosis has to be based rather on an aggregate of small points than on any one symptom. The subjects of disseminated sclerosis are as a rule younger, usually between 20 and 30 rather than between 30 and 40, as in the case of subacute combined degeneration, remission of symptoms frequently occurs, nystagmus is more pronounced, and the sphincters are usually affected early. On the other hand, the slight affection of the arms while the legs are markedly affected, the presence of girdle and lightning pains, and the symmetrical affection of all four limbs, are points in favour of subacute combined degeneration.

In distinguishing the disease from peripheral neuritis it is only during the third stage that any difficulty is liable to arise, and then apart from a definite history of a spastic stage, the incontinence of urine, the anæsthesia of the trunk, the girdle pain, and the extensor response of the plantar reflex are sufficient to exclude the peripheral affection.

The diagnosis from acute myelitis is by no means easy in the absence of a distinct history of a gradual onset. A rapid onset of symptoms with early occurrence of bladder trouble, marked pain and tenderness in the back on bending the head forward, absence of any affection of the arms, greater disturbance of temperature, more rapid wasting of muscles, and complete loss of reflexes, would all be in favour of myelitis. Nevertheless, certain cases of acute myelitis very closely resemble those of combined degeneration of the cord.

A tumour involving the spinal cord and giving rise at first to a spastic paraplegia, followed by flaccid paralysis, might suggest the possibility of a combined degeneration of the cord, but the presence of severe radiating root pain, complete absence of any affection of the arms, and the early onset of bladder symptoms, would all be in favour of the diagnosis of tumour.

*Prognosis.*—The disease in most cases runs a steadily progressive course from its onset to its termination in two to three years; in certain cases, however, it has run through its various stages in less than six months, improvement occasionally takes place, but is not as a rule long maintained.

*Treatment.*—Treatment has almost no influence on the course of the disease, except in so far as the anæmia is concerned, which will often improve on the administration of arsenic and iron. Bedsores are almost certain to form during the third stage of the disease in spite of the most careful nursing. Cystitis, which not infrequently occurs, is not as a rule severe, and yields to the ordinary treatment.

*LITERATURE.*—The bibliography of the subject will be found in a paper on "Subacute Degeneration of the Spinal Cord," *Brain*, vol. xxiii. p. 108.

## II. FLACCID PARALYSIS

*FAMILY PERIODIC PARALYSIS.*—*Definition.*—A disease occurring in families and characterised by attacks of widespread flaccid paralysis, with loss of reflexes and electrical excitability of the muscles, without sensory, vesical, rectal, or psychic disturbance, and with intervals of perfect health.

*Historical.*—The first case of this disease was described by Cavaré in 1853 in a woman aged 24, who had several attacks of generalised paralysis. In 1876 Hartwig published a case of intermittent spinal paralysis in a patient aged 23. In 1885 Westphal brought the condition prominently forward in a paper "On an unusual Case of Periodic Paralysis of all four Extremities with simultaneous Loss of the Electrical Excitability during the Paralysis." The case was that of a boy aged 12 years, in whom the attacks



began with weakness in the legs; after a short time the arms and legs were completely paralysed and flaccid, the knee-jerks were not obtainable, and the electrical examination showed quantitative diminution to both galvanism and faradism. The following day improvement took place, and gradually became complete. On this case he says: "This relative rapid loss and return of electrical irritability in the nerves and muscles is without parallel. We know neither a disease of the spinal cord nor of the nerves in which anything of the like nature has been observed; even physiology fails to explain such a condition."

Then followed a series of papers by Goldflam, Oppenheimer, and others, while in 1898 Taylor of Harvard published a case of a boy in whose family the disease could be traced for five generations. To this is appended an admirable digest of the subject up to that date.

*Etiology.*—The age at which the disease most frequently occurs is between the second and third decade of life, but a few cases occur during the first decade, and also during the later periods of life. It would seem probable, therefore, that the affection owes its origin to certain conditions prevalent in youth.

Both sexes are equally affected, and the malady is transmitted through the female as well as through the male line.

The distinct disposition of the disease to affect various members of a family, and to pass from parent to child, is abundantly proved by the published cases, for out of 54 cases recorded 35 occurred in three families, while the remaining 19 were either sporadic or associated with one other case in the same family. On the other hand, the families in which the disease occurs are not of a degenerate type, nor unusually subject to neuroses; in fact, Taylor states that the disease occurs in families of unusual nervous stability. In Craft's case, however, there was a distinct neuropathic family history.

The periodicity of the disease, the fact that malaria has been present in certain cases, and that the attacks have yielded to quinine, have led some observers to suggest that the condition is malarial in origin. There is, however, little evidence to show that the association of the two affections has been more than a coincidence, and the plasmodium has never been found in the blood from a case of periodic paralysis. The attacks often follow on muscular exertion, though this is by no means always the case, for some patients have attacks more frequently while at rest than when at work.

In some cases certain articles of diet seem to have had the effect of producing an attack. Certain peculiarities in the physical development have been noted, and among these a condition of muscular hypertrophy, which did not correspond with the muscular strength.

*Symptoms.*—The following may be taken as a general description of an attack which commonly commences at night. If the patient is awake a feeling of weakness is experienced in the limbs. The loss of power begins in the proximal muscles of the legs, gradually passes to those more distal, and then affects the abdominal and intercostal muscles; but before the paralysis in these becomes complete it involves the proximal muscles of the arms, and then the distal muscles, the limbs becoming absolutely flaccid and powerless, and respiration being carried on by the diaphragm alone. Occasionally the face may be affected, and some of the other cranial nerves may be involved, but in most of the described cases the muscles of the face and jaw have escaped. There is no difficulty in articulation, and there is no incontinence of urine or feces. There is no mental disturbance and no

pain. The appetite is often lost, but there is no digestive disturbance, nor is there any difficulty in swallowing, nor does the patient experience dyspnoea. After the attack has reached its maximum, which it usually does in three to four hours, recovery begins to take place slowly, following the reverse order to that of the onset of the paralysis, *i.e.* the legs are the last to recover. The period of recovery is about the same length as the period of onset. In other cases the duration of the whole attack is longer, and in Taylor's case the patient who awoke paralysed in the morning did not begin to recover till the afternoon of the same day, and recovery was only complete on the following morning.

*Reflexes.*—During the most profound stage of the attack all the deep reflexes are lost, and return as the motor power is restored to the limbs. The superficial reflexes are also generally lost, but in some of the reported cases the abdominal and plantar reflexes have been present. In the intervals of health the reflexes are present and normal.

*Electrical Changes.*—One of the most striking features of this disease is the loss of electrical excitability, which is to be noted on the examination of the muscles. In the free intervals the muscles have generally been found to give a quick and normal response both to the faradic and galvanic current. Both Goldflam and Taylor have, however, found diminished excitability in certain muscles even during the free interval, and also that the muscles undergo rapid fatigue.

At the height of an attack there is complete loss of reaction, both to faradic and galvanic stimulation, and as recovery of motor power takes place the reaction to faradism and galvanism returns. A quantitative diminution in the reaction of the muscles of the face has also been observed.

The muscles lose their mechanical irritability during the height of the attack, while they respond to a like stimulus during the free interval.

Sensation remains perfectly normal during an attack, and there is no vaso-motor disturbance.

The rectal and vesical functions remain normal, there is neither incontinence nor retention of urine, although the patient may not micturate for thirty-six hours. This lack of desire to micturate is probably due to diminished secretion, as there is no distension of the bladder. The bowels do not act during an attack.

The temperature is practically unaffected during an attack.

The pulse and respiration may both be slightly increased in rate, but not to any marked degree.

Nothing abnormal can be detected in the lungs. In the heart, however, it has been noted that there is slight enlargement during an attack, and that a murmur indicative of mitral regurgitation becomes audible. As the patient recovers this passes off. No temporary enlargement of the spleen or liver has been detected, and there is no intestinal distension.

The examination of the blood during an attack shows a marked lymphocytosis, which in Taylor's case was present also during the free intervals. Lymphocytosis is said to be normal for a child of 10, but not for a man of 20.

The examination of the urine has shown nothing abnormal, although in Craft's case more detailed examination was made, and an increase of the ethereal sulphates found. He considers that this is what might be expected from the breaking up of toxic materials in the intestines and their elimination as sulphates. An ethereal extract was isolated from the stools in this case, and 1 gramme injected into rabbits and guinea-pigs produced a paralysis which gradually disappeared in forty-eight hours. The nature of this poison has not been ascertained.



*Duration and Frequency of Attacks.*—The length of the attacks varies considerably, but may be said to last between ten and forty-eight hours.

The frequency of the attacks also varies greatly, and at times they may occur almost daily, in other cases they may be repeated every week or month, while in others there may not be more than one attack a year.

Patients with this affection are probably subject to attacks during the whole of their life; on the other hand, in Taylor's family the attacks were said to grow less frequent as life advanced, and finally to have disappeared entirely between 40 and 50.

*Morbid Anatomy.*—No post-mortem has been made on any one of these cases, and it is hardly to be expected that any change gross enough to be visible by the present microscopical methods would be found. Small pieces of muscle have been removed and examined microscopically, and increased diameter of individual fibres, vacuolation, and waxy degeneration have been found.

*Pathology.*—The pathology of the disease is most obscure, as Westphal says: "We know neither a disease of the spinal cord nor of the spinal nerves in which anything of like nature has been observed." It has been suggested that a poison similar in action to curare is formed within the body, but even in curare poisoning the muscle retains its mechanical irritability. Even if one accepts the theory that the condition is produced by some autotoxin, such as has been found to be present in the stools by Craft, and on injection into animals gave rise to a transient paralysis, one is still in a difficult position to explain on what part of the nervous or muscular system such a poison acts. It can hardly be a general muscular poison acting directly on the muscles, for then one would not expect the muscles of the face to escape, and again the onset of the paralysis and its ascending character is strikingly like that seen in Landry's paralysis. On the other hand, if the toxin acts on the lower motor neurone, it is impossible to conceive how such a lesion could give rise so rapidly to such a profound change in the muscles that they should fail to respond both to a faradic and galvanic stimulus. The same objection applies to a periodic vascular disturbance of the cord, such as has been suggested by Westphal. If then one supposed the condition to be dependent on an autotoxin, the poison must be regarded as acting not only on the lower neurone, but also on the muscle directly; whether the vascular condition of the cord has any influence on the selective action which the toxin seems to exert must remain doubtful, but it seems not improbable that such a factor may, however, exert some such influence.

*Diagnosis.*—In a case seen for the first time, and without a definite history of previous attacks, the diagnosis of Landry's paralysis might with justice be made; on the other hand, when the disease occurs in a typical form with a history of previous attacks the diagnosis presents no difficulty.

Some authors have allied the condition with the dystrophies, and the change found in the muscles and the permanent weakness of some groups of muscles as described in certain cases would seem to support such a suggestion. The course of the disease is, however, entirely against such a view.

It has also been suggested that it is a condition allied to congenital myotonia (Thomsen's disease, see vol. viii. p. 189).

*Prognosis.*—The disease seems to have no tendency to diminish the duration of life. Most patients seem to be liable to attacks during the whole of their life, but in others a tendency to recover has been noted about middle life.

*Treatment.*—No treatment is known which will produce any result. Quinine has been given in some cases with good effect, but in others it has had no influence.

LITERATURE.—TAYLOR, E. WYLLYS. *Journal of Nervous and Mental Diseases*, 1898.—CRAFTS. *The American Journal of the Medical Sciences*, 1900.—SINGER and GOODEBODY. *Brain*, 1901.

LANDRY'S PARALYSIS.—Many writers now include this affection, otherwise known as "acute ascending paralysis," in their accounts of peripheral neuritis, from which it is undoubtedly difficult to distinguish some of the cases. In like manner there may be a close resemblance to acute poliomyelitis; but the clinical picture in other cases is so different from that of the ordinary forms of either of these affections, that until we have more proof that all the cases can be accounted for by peripheral neuritis or poliomyelitis, it is well to give this symptom-complex a separate description.

*Definition.*—The characteristics of the malady have been supposed to be rapidly progressive motor paralysis, which usually commences in the lower extremities and mounts upwards, and which, when it reaches the muscles of respiration, causes death by asphyxia; the muscles preserve their nutrition, and respond normally on electrical stimulation, and cutaneous sensibility and the sphincters remain intact.

*Etiology.*—Men are attacked a good deal more frequently than women, the relative proportion being about three to one. Although most common between the ages of 20 and 40, it oversteps these limits, and is met with both in older people and in children. In some cases no cause can be discovered, while in others there has been reason to suspect toxic agents, such as are known to produce multiple neuritis. Not uncommonly, one of the acute specific fevers has been the immediate precursor, and septicæmia, the puerperal state, and syphilis, are among the known antecedents. The malady has followed severe exposure to cold in some cases, and it sometimes develops in persons addicted to alcoholic excess.

*Morbid Anatomy.*—The disease may prove so rapidly fatal that it is not surprising that no anatomical lesions have been found to account for the clinical symptoms in some cases. Most of the cases of this kind are, however, of no value, as they were examined at a time when the histological technique had not reached its present state of perfection. Nevertheless, no changes have been found in the peripheral or central nervous system in a few of the cases that have been examined by the aid of recent methods. In the great majority, however, definite changes of a degenerative character have been detected in some part of the peripheral motor neurones. Either the cells are affected in the anterior horns of the spinal cord, or nuclei of cranial nerves, or the axones in the anterior roots, or peripheral, spinal, or cranial nerves, show changes, or it may be that the whole peripheral neurone is altered. In addition to the degenerative changes in the nerve elements, vascular changes may be present, and consist in engorgement of vessels, perivascular exudation, and minute hæmorrhages.

Bacteriological examinations have been made in several cases; in some with negative results, while in others the information derived cannot be regarded as definitely establishing a causal connection between the micro-organisms found and the morbid changes present; moreover, the same microbe has not been found in all cases.

*Pathology.*—The sudden onset and rapid evolution of the disease strongly suggest that the action of a toxic agent is responsible for the clinical mani-



festations, while the absence of anatomical lesions, in some cases, and the complete recovery of other patients, also support this view. Moreover, such changes as have been discovered in the central and peripheral nervous systems are such as commonly result from the action of known poisons such as alcohol and the like. There is nothing to suggest that the same poison is in action in all cases, and the meagre results of bacteriological examination do not allow of the conclusion that the poison is of bacterial origin. Nevertheless, the circumstances under which many of the cases arise are strongly suggestive of this mode of origin of the disease in them, though in other cases known poisons of a different nature have existed, and may have caused the malady. In yet another group of cases the circumstances under which the affection occurs give us no clue as to the possible nature and source of the poison. If a toxic agent does produce the disease, it behaves very much like lead, for it exercises a similar selective action in that it almost exclusively attacks the motor neurones.

*Symptoms.*—The paralysis may be abrupt in its onset, or there may be premonitory symptoms lasting for some hours or days. In the majority of cases in which they occur the premonitory symptoms consist in subjective sensations such as numbness, tingling, formication, and the like, which especially affect the hands and feet; or vague, rheumatic-like pains may be felt about the trunk and in the limbs. In other cases the pains are sharper, and tend to shoot along the course of the nerves, and there may even be tenderness of the muscles of the calves. With or without such sensory disturbances there may be a feeling of lassitude and general weakness, or it is noticed that a short walk induces unusual and undue fatigue, so that the legs perhaps feel heavy. In some cases there has been sweating of the feet and hands, or the extremities have been cold or even livid.

Whether ushered in by these premonitory symptoms or not, the most prominent symptom of the actual attack is motor paralysis, which usually commences in the lower extremities, and one limb commonly becomes weak before the other. Standing and walking quickly become impossible, and in a few hours or days all movements of the lower extremities, even as the patient lies in bed, are impossible; the limbs lie like helpless logs, and there is marked dropped foot on both sides. The trunk muscles next become involved, so that the patient is unable to rise into the sitting posture or even to turn over in bed. Now, or even sooner, the arms begin to become weak; a certain amount of clumsiness in using the fingers is the first defect noted, but the weakness may spread up the limbs until they become as completely paralysed as the lower extremities. Indeed, in some cases, otherwise typical of the class under consideration, the weakness has commenced in the upper limbs, and the lower extremities have subsequently been attacked. The neck muscles become weak in some cases, so that the patient cannot turn the head from side to side, or raise it from the pillow. The intercostal muscles become weak, and respiration largely diaphragmatic; but the diaphragm may become paralysed before the intercostals. When the diaphragm and intercostals are both paralysed there may still be a brief struggle for life, owing to the action of the extraordinary muscles of respiration.

Paralysis in the distribution of some of the motor cranial nerves occurs in a considerable proportion of cases, and the most common defect noticed is some articulatory difficulty of speech owing to weakness of the lips and tongue, and the speech may have a nasal quality owing to weakness of the palate. Difficulty in swallowing is also a common symptom, and may be so pronounced that the patient has to be fed by the stomach tube. All



degrees of defect of phonation have been described up to complete aphonia, but most often there is only slight weakness of the voice, which may be accounted for by weakness of the muscles of respiration, or the voice may be hoarse or squeaky owing to weakness of the laryngeal muscles. Paralysis of the facial muscles on one or both sides, weakness of the muscles of mastication, and even affection of the ocular muscles, with ptosis and strabismus, occur, and the pupils may be unequal and defective in their action.

The paralysis is unaccompanied by any spasticity in the muscles; on the contrary there is the most absolute flaccidity. Such patients may die while their muscles are still well nourished, and at a time when the electrical reactions are normal. This, however, entirely depends on the length of time that has elapsed between the onset of the illness and the time of death—a period that is insufficient, in most cases, to allow the muscles to waste appreciably, or respond in an abnormal manner on electrical excitation. When the patients do not die, or when they recover partially or completely, some of the affected muscles waste, and show the partial or complete reaction of degeneration.

With the onset of flaccid paralysis the tendon jerks are rapidly abolished, and in the fatal cases the knee-jerks remain absent up to the time of death. With recovery, however, all the tendon jerks return, though the knee-jerks as a rule remain absent for a long time.

The sphincters usually escape entirely, and in some cases in which there appears to be retention of urine this is probably due to defect in expulsive power consequent on weakness of the abdominal muscles, which also, no doubt, accounts in some measure for the obstinate constipation that may be present. In quite exceptional cases paralysis of both sphincters, and in consequence incontinence of urine and fæces, has been observed.

In the large majority of cases there are no sensory defects other than the subjective sensations that may occur as premonitions of the attack, and which may persist after definite motor paralysis has supervened. The most delicate methods of testing sensibility usually fail to determine any anæsthesia; but in a few cases that appear to belong to the class under consideration, slight blunting of sensibility has been detected, while in some anæsthesia of a more pronounced character has been described. Hyperæsthesia of the skin and muscles has also been occasionally met with, and even definite tenderness along the course of the nerve trunks has been observed.

The patient usually remains absolutely clear in intellect, so that it is exceptional to meet with psychical disturbance.

Whether the onset of the malady is attended with rise of temperature or not, there is always some febrile disturbance in the final stages which immediately precede death, at which time there is also increased frequency of pulse. The urine occasionally contains albumin. The spleen is enlarged in some cases, and this has been regarded as in favour of the view that the disease is toxic in origin.

*Diagnosis: Polyneuritis.*—As the resemblance between acute ascending paralysis and polyneuritis is sufficiently close to lead some observers to consider that the affections are identical, it naturally follows that the possibility of distinguishing the one from the other is not always easy, and it becomes almost impossible if we are to admit that cases in which there is blunting of sensibility may, nevertheless, belong to the former class. The presence of definite anæsthesia, and the fact that the paralysis attacks the superior as well as the inferior extremities, and spreads up the limbs to the



trunk, instead of passing through the trunk before the upper limbs become affected, are the chief points that help us in arriving at a decision.

*Poliomyelitis*, as usually met with, is random in distribution, and does not preserve the symmetry observed in acute ascending paralysis; nevertheless, some of the cases that present all the features of the latter affection in their earlier stages are subsequently left much in the same condition as is seen in severe and extensive poliomyelitis.

*Myelitis*.—In ascending myelitis all the functions of the cord suffer, so that in addition to the motor paralysis there is marked blunting of sensibility, a great tendency to trophic disturbances in the skin, and loss of control over the sphincters.

*Meningeal hæmorrhage* may cause ascending paralysis, but muscular spasm is a prominent feature, and the pain in the back is much more severe than anything experienced in Landry's paralysis.

*Prognosis*.—The majority of the patients die from asphyxia consequent on paralysis of the muscles of respiration, or death may be due to cardiac failure when the disease attacks the medulla oblongata. A fatal termination is usually reached in about a week from the commencement of the illness, but some patients die within forty-eight hours. Less commonly the fatal issue is delayed until the end of the second, or it may even be the third or fourth week. Paralysis of the muscles of respiration or disturbed heart's action are symptoms of most serious import, as are any signs of bulbar affection. In some cases the disease becomes arrested and ultimately terminates in complete recovery, despite the fact that the arrest may not take place in some until the respiratory muscles are so weak that there is serious embarrassment of breathing, and the patient appears to be in great jeopardy. Convalescence may only last a few weeks, or it may be prolonged to several months. In other cases, although the disease becomes arrested, there is unfortunately little or no recovery, and most if not all of the affected muscles remain permanently paralysed or undergo atrophy.

The forecast depends on what stage the illness has reached before the patient is seen, and on whether the disease has become arrested or is still progressing. Prognosis as regards life depends on the degree of affection of the muscles of respiration, and on whether there is disturbed heart's action, or other evidence that the morbid process has attacked the medulla oblongata. Moreover, when the case is seen in the early stages a guarded prognosis must be given, for any of these untoward symptoms may, of course, become manifest later. The possibility of recovery from the paralysis can only be estimated after the disease has become arrested, and a favourable opinion is then only justified when there are distinct signs of improvement within at least a few weeks of the arrest, for the outlook is hopeless when several months have elapsed without any, or but slight signs of recovery from the paralysis.

*Treatment*.—If the case is seen early, mercurial inunction ought to be commenced at once, whether there is a syphilitic history or not, for this drug may reasonably be expected to influence other toxic blood states as well as that due to syphilis. Strychnia should be administered by subcutaneous injection—a procedure that becomes imperative on the appearance of any signs of weakness of the muscles of respiration. Ergotin has been recommended, and one patient improved so much while taking this drug that it deserves a trial in other cases. Hot or vapour baths have been recommended at the outset in cases that follow exposure to cold, and counter-irritation to the spine has also been supposed to be of service. The patient's strength has to be maintained by nutritious food, but care must be



observed in its administration when there is difficulty of swallowing, so as to avoid choking or the impaction of food in the larynx. The difficulty in swallowing may be so great as to necessitate the use of the nasal tube. Care is needed not to overload the stomach, or induce flatulent distension, for either may seriously embarrass breathing when the muscles of respiration are weak. If the patient survive the acuter stages of the attack, all our efforts must afterwards be directed to keep up the nutrition by good food and cod-liver oil and malt preparations, and to prevent him from taking cold, for bronchitis and pneumonia readily prove fatal in consequence of the weakness of the muscles of respiration. The use of strychnia must be continued, though it may now be given by the mouth, and other tonics, such as iron, phosphorus, quinine, and arsenic, may be substituted from time to time. Local measures are of great value at this stage of the illness, for by massage and galvanism we may hope to improve the nutrition of the muscles that have already wasted, and to lessen the degree of atrophy in cases that are seen before this has become a prominent feature of the paralysis.

### III. PARALYSIS WITH ATROPHY OF MUSCLES

(See also "MUSCLES, DISEASES OF," Vol. viii.)

**ACUTE ANTERIOR POLIOMYELITIS.**—*Definition.*—A disease of acute or sudden onset, occurring commonly in young children, and characterised by loss of power and usually subsequent wasting of the part affected, without loss of sensation or affection of sphincters.

*Etiology: Age.*—By far the greater number of cases occur during the first three years of life, the greatest number during the second year. After the end of the third year the disease becomes less frequent, and after the age of six it may be considered rare; cases, however, occur both in adult life and old age.

*Sex.*—This would seem to have no influence, since the disease occurs in about an equal number of cases in the two sexes.

*Seasonal Relationship.*—That the disease is dependent on the season of the year is clearly shown by an analysis of the time of onset in a series of cases. The period of year in which the onset is most common is during the months of July and August. Starting from January, the curve remains low till the end of May or the beginning of June, when it rapidly rises, reaching its maximum in August, and then somewhat suddenly falls to its mean in November. The period of onset thus corresponds with the months of greatest heat in England.

*Other Etiological Factors.*—The relation of the disease to the acute specific fevers is one of extreme importance. It has been observed to follow scarlet fever, measles, whooping-cough, typhoid fever, pneumonia, diphtheria, and malaria, and the relationship has been too frequent to allow of doubt that there is some significance in the connection. Exposure to cold and damp are not infrequently the assigned causes, and in view of the pathology of the affection may certainly be regarded as important factors. Some cases have been noted to follow injury.

The occurrence of the disease in an epidemic form is definitely established, since well-marked epidemics have been reported in various countries and in parts of the country which at other times have been perfectly free from the malady.

In relation to this epidemic form must be noted the occurrence of febrile attacks and other forms of paralysis in members of the same family at or about the same time. A very interesting series of cases of this nature were



recorded by Dr. Pasteur, in which two members of the family had definite anterior poliomyelitis, a third had an attack of hemiplegia, two others had febrile attacks without any paralysis, and two more had certain nervous manifestations, which rapidly passed off in a few days; all these children were taken ill within a few days of one another. There is in nearly every case some infection, but the nature of that infection is not always the same; just as thrombosis is liable to occur after many different forms of infection, so infantile paralysis may follow infective processes of entirely different nature. This point will, however, be dealt with more in detail in the discussion of the pathology of the disease.

*Symptoms: Acute Stage.*—The onset of the disease is sudden. Sometimes the child goes to bed well and wakes at night screaming with pain, and if the little patient is old enough it may be able to indicate that the pain is in one or other limb, or in the back. The child resents all movements of the limb. The temperature is raised, vomiting may occur, there is general malaise, and the onset may even be accompanied by convulsions. Possibly the more common history is that the child is taken ill with slight febrile symptoms, there is headache, vomiting, possibly some diarrhoea, and pain in the back; the child is kept in bed for a day or so, and then it is noticed that one or more limbs are paralysed.

Another form of onset is described by some authors in which, without any disturbance of health, there is sudden and complete loss of power in one or more limbs, the temperature is not raised, and there appears to be no pain. Such an onset would seem to us to be of greater rarity than has been stated by some, viz. 25 per cent.

In some cases there is a sudden onset of paralysis which may persist for a few days, and which then rather rapidly clears up so as to leave no trace of weakness.

In other cases the onset has been more gradual, so that the height of the paralysis is not reached for two to three weeks, while in others again there is the sudden onset followed by some improvement, and then after some days an extension of the paralysis.

These last two forms are more frequently seen in the adult than in the child.

*Temperature.*—The temperature generally rises rapidly to between  $102^{\circ}$  and  $103^{\circ}$ ; it remains high for a day or two, and then falls in about a week to the normal. This is by no means always the case, for sometimes the temperature will remain elevated for three to four weeks, especially if the limb continues to be painful on movement.

Pain is commonly present during the onset of the disease; it is as a rule referred to the limb affected, and rapidly passes off, so that in a few days' time the leg can be passively moved in all directions without pain.

Sometimes the pain is referred to the neck, and may occasion some rigidity of the muscles in this region. In other cases pain on movement persists for a considerable time, often for some three to four weeks.

*Sensation.*—Although pain may be referred to the limb, yet on examination no alteration in tactile or painful impressions can be made out. Cases have been recorded in which such alterations have been present, but they are uncommon, and there is no doubt that in the majority of cases no such alteration exists even during the acute stage.

*Loss of Power.*—The extent of the paralysis usually reaches its maximum in twenty-four to forty-eight hours from the onset of the illness, in some cases within a few hours. At such a period the child may be paralysed in all four extremities, and also in the thoracic and abdominal muscles,

respiration being carried on by the diaphragm alone; on the other hand, it may be limited to one limb. The amount of the paralysis during the acute stage is always in excess of that at a later stage, so that a child who is paralysed in all four extremities during the acute stage may possibly at a later stage be only completely paralysed in one leg, with some weakness of the opposite limb, the arms having completely recovered,—in fact they are often extremely well developed owing to their use for progression.

The paralysis rarely extends to the cranial nuclei, but affection of the muscles of the face does sometimes occur. The sphincters also are rarely affected, but when the lesion is extensive and involves the lower sacral region both the sphincters may be affected.

The character of the paralysis is always flaccid, and there is a complete absence of tonus in the muscles affected; it may be noted, however, that there is sometimes a certain amount of rigidity in groups of muscles represented in segments adjacent to those actually involved.

*Reflexes.*—During this acute stage all the reflexes, both superficial and deep, are abolished in those parts of the body that are affected.

The second stage of the disease is reached when the acute febrile state has passed and the transient paralysis, which may have affected all four extremities, has cleared up, leaving certain muscles of one or more limbs paralysed. Wasting of the muscles now generally becomes manifest, but it should be remembered that the atrophy which they undergo is not always obvious, and that in infants who are fat very little difference in measurement can be detected between the two limbs, although the one is flaccid, and the other is firm and is moved normally. Owing to the complete loss of tone in the muscles, the limb often hangs in a pendulum-like manner, and swings to and fro as the position of the body is altered.

On electrical examination of the muscles at this stage it is found that they give the reaction of degeneration, *i.e.* they react very slightly, if at all, to faradic stimulation, and briskly to a galvanic current, the ACC being greater than the KCC. Fibrillary tremors are occasionally to be noted in the muscles, but they are not common.

In reference to the statement made above that the atrophy of the muscle is not always obvious, it is important to bear in mind that at the time of life when a child begins to lose its excessive fat it is noticed that the leg is wasting, and therefore the disease is considered to be progressive, whereas this is not really the case.

The temperature of the affected limb is markedly lowered even during the early stages of the disease; the limb not only feels cold and is of a blue colour, but the actual temperature is some degrees below that of the normal limb.

During this stage, although atrophy of the muscles is taking place, return of power is also in progress. Those muscle fibres that were represented in the cord by cells which have been completely destroyed, undergo atrophy, whereas other cells to which the damage was slighter recover, and the muscle fibres in connection with them also recover.

In some cases the recovery of muscular power is comparatively rapid, and in a month or so there is good return of power; in others it is a much more gradual process, and may be prolonged over a year or more.

It is during this stage that contracture takes place, and the factor that chiefly influences the production of such deformities is the extent of implication of any group of muscles; for instance, if all the muscles below the knee are completely paralysed very little deformity takes place, but if the anterior tibial group are considerably affected (as not infrequently happens) while



the gastrocnemius is but little affected, then the foot tends to assume the position of talipes equinus; on the other hand, if the gastrocnemius is affected while the anterior tibial group are not paralysed, then there results a position of talipes calcaneus.

The knee-jerk is absent or present according to whether the quadriceps, and especially the vastus internus muscle, is or is not involved.

The third stage of the disease is reached when there is neither further atrophy nor further recovery of power. The growth of the limb is impaired, so that as the child grows the affected limb becomes relatively shorter than the sound limb. The bones are thinner, the skin is soft and loose on the subcutaneous tissue, but the hair is normal, and it is stated that the erector muscles of the hair are also unaffected. Sometimes the growth of the limb is so much inhibited that it remains in a condition of complete atrophy, and as the individual grows it hangs down as an undeveloped appendage from the trunk.

Owing to the shortening of one extremity it not infrequently happens that a condition of lateral curvature of the spine is produced, the pelvis becoming tilted so as to counteract the shortening of the limb.

Following on the flaccidity of the muscles certain changes take place in the joints, the ligaments stretch, and the joint tends to fall away from its socket; this is especially well seen in the case of the shoulder joint, and the head of the humerus can be felt to hang below the glenoid cavity.

In the case of the knee joint the stretching of the ligaments gives rise to a condition of genu recurvatum or genu valgum. If this condition is allowed to persist, changes take place in the surface of the joint so that they adapt themselves to their new position.

In this third stage the electrical excitability is so diminished in the paralysed muscles that they will no longer react either to faradism or to galvanism.

The disease now remains stationary for years, and it may be for life; on the other hand, it must be noted that in a certain number of cases after the disease has been stationary for years progressive muscular atrophy begins to manifest itself, either in the limb originally affected or in one of the unaffected limbs.

*Distribution of the Paralysis.*—It is stated that the disease may give rise to paralysis of almost any muscle of the body, on the other hand the seat of election is undoubtedly the muscles of the leg. Out of 595 cases of the disease collected by Allen Starr, in no less than 516 were one or both legs affected, *i.e.* 90 per cent. The muscles in the leg most commonly affected are the anterior tibial group; those least commonly affected in the limbs are the small muscles of the hands.

*The Production of Deformity.*—The cause of the production of deformity has been a matter of some dispute, it is generally ascribed to the tonus which is present in the unaffected muscles; but some authors attribute it to the force of gravity, together with the organic contraction which occurs in the affected muscles, and to the lack of development in the muscles and tendons while the osseous structures continue to grow in length.

There is no doubt that all these factors have their influence, but in the deformities produced in the earlier stages of the disease it is certain that the most important factor is the effect of "tonus" in the normal or only partially paralysed muscles. This fact is well illustrated in the contrast there is between a case in which all the muscles below the knee are paralysed, and one in which only one group is affected. In the former case there is simply flaccidity without deformity, while in the latter there is

well-marked talipes, equinus, or talipes calcaneus, according to which group of muscles is paralysed. It is the occurrence of this form of deformity that passive movement and massage will do most to remedy. In the later stages of the disease the organic changes which take place in the muscles, together with the other factors above mentioned, and the constant erroneous position of the limb, have most to do with the production of the deformities that occur.

The lateral curvature of the spine may in the same way during the earlier stages of the disease be due to the unopposed action of the erector spinæ, while in the later cases it is more commonly produced by the tilting of the pelvis in order to compensate for the shortened leg on the affected side.

*Pathological Anatomy.*—As in the case of the clinical symptoms, so with the pathological anatomy, three stages can be recognised as a matter of convenience. If an opportunity arises for an examination during the acute stage nothing abnormal can be seen on the surface of the cord or its membranes; on section, however, the gray matter of the anterior horns appears of a deep red colour and softened, and it may be so engorged as to appear as if hæmorrhage had taken place into the gray matter. The focus of destruction is generally most acute at one spot, commonly situated in the lumbar region, and from this point the congestion extends for a variable distance, only the most acute part of the process being visible to the naked eye, although when examined microscopically it is seen that the process has a far wider distribution, and that the vessels of the anterior horns of the affected parts are engorged with blood and surrounded by a large number of inflammatory exudation cells. At other points minute extravasations of blood occur. In the regions most affected few if any nerve cells are to be seen, while at other points where the process has been less severe, swollen and altered nerve cells are present. There is, in fact, an inflammatory condition of that part of the gray matter of the anterior horns which is supplied by the branches of the artery passing down in the anterior fissure of the cord.

In the second stage, after the acute inflammatory condition has subsided, the gray matter of the anterior horns has a translucent appearance, and it is often so soft that on section after the cord has been hardened, a depression occurs in the situation of the anterior horn, giving rise to the appearance of a small cavity.

If this condition is examined under the microscope it is found that the hyaline material is composed of very fine fibrils, in which there are numerous newly-formed vessels. In the whole of the softened area there are abundant fat granules, and these are especially numerous in the perivascular lymphatics, so that all the larger vessels are lined out by the presence of these fat globules. In the part most affected no trace either of nerve cell, nor of any of the fine medullated fibres, which are normally present, can be seen, although in parts less affected both normal cells and fibres are present. Although the process is so strictly limited to the immediate region of the anterior horn, yet numerous degenerate fibres can be found scattered throughout the antero-lateral tracts of the cord, and also to a lesser extent in the posterior columns; these fibres are probably endogenous in origin. Occasionally, also, when there has been damage in the region of Clarke's column, in the lower dorsal region, degenerate fibres are found in the direct cerebellar tracts.

Degeneration can also be shown to be present in the anterior roots.

In the spinal cord from a case of several years' standing it is seen that



the anterior horn of the affected side is reduced in size, and also that the lateral columns are smaller than the corresponding columns of the opposite side. Under the microscope it is seen that the vessels of the affected area are thickened, there is a complete absence of nerve cells in the anterior horns, and the whole interior horn is filled up with connective tissue. In other parts where the process has been less acute a few normal ganglion cells may remain together with others that are atrophied and shrunk. Many of the fine medullated fibres which usually form part of the anterior horn have been destroyed, and the contrast in this respect between the posterior and anterior horns, or between the two anterior horns, in those cases in which the condition is unilateral is striking. The anterior roots are atrophied.

The peripheral nerves during the acute stage show some degeneration, which affects for the most part the smaller medullated fibres; in the later stage of the disease these fibres undergo atrophy, and all that is to be noticed about the nerve is that it is somewhat smaller than normal, with possibly some increase of the connective tissue between the fibres. It is very remarkable to notice the number of normal medullated fibres which supply a completely atrophied muscle; these fibres have been shown to arise from the posterior roots, and to supply the muscle-spindles and the musculo-tendon organs within the muscle.

*Muscles.*—The muscles that are affected undergo atrophy, the individual muscle fibres gradually become smaller and smaller, the transverse striation is retained even when they are atrophied to an extreme degree.

Fatty degeneration of the muscle fibre does not usually occur, although the final condition of the muscle may be such that it is composed entirely of fat; this fat becomes deposited between the atrophied fibres, and not in them. Not infrequently small areas of muscle retain their normal development. In a muscle which has undergone almost complete degeneration several muscle-spindles can be seen, these contain one or more perfectly normal muscle fibres with normal transverse striation, and in suitably stained specimens it can be shown that the nerve fibres supplying these spindles are perfectly normal.

In some cases the muscle, instead of becoming fatty, is found to be sclerosed, that is to say, the muscle fibres undergo atrophy, connective tissue increases between the fibres, and, finally, the muscle becomes simply a mass of connective tissue in which, however, on section the normal muscle-spindles can still be shown to be present.

*Pathology.*—All observers will agree that the vessels in the affected area are thrombosed, and that there are signs of inflammatory reaction around them. But the question whether such thrombosis is a primary or secondary condition is one on which opinions differ. In considering this question it should be borne in mind that the disease is of greatest frequency during the hot period of the year, that it occurs in epidemics, and after the acute specific fevers. These various factors suggest that the primary cause of the disease is not common to all cases, but that the various causes, be they the exposure to chill in hot weather, or bacterial or other infection, produce a morbid blood state which is the cause of the disease. The view that is most widely accepted nowadays is that the condition is infective in origin, and the changes that result are inflammatory in character. There is, however, much to be said in favour of the view that the process is one depending on a primary thrombosis of the vessels, and in support of this statement may be cited the rapid onset of the paralysis simulating a vascular rather than an inflammatory lesion, the limitation of the destructive process to the area



of the cord supplied by a branch or branches of the artery of the anterior median fissure, and that both the acute condition with multiple extravasations of blood and the subsequent softening simulate the condition which is seen in the brain after thrombosis of the smaller vessels.

The wide distribution of the paralysis at the onset is easily accounted for by the vascular disturbance taking place in the artery of the anterior median fissure which supplies the whole of the anterior horns, and the fact that smaller areas of thrombosis are found at various levels is only what might be expected as the result of the retardation of the blood stream in the vessel. With a vascular theory to explain the condition, it is not difficult to see why the lumbar region of the cord should be more frequently affected than the cervical or dorsal, for it must be noted that the lumbar region lies at a point most distal from the blood-supply, and also it has been shown by Moxon that this part of the cord fails to become injected experimentally through the reinforcing arteries which pass up in the roots.

Bacteriology has yet to prove that there is one specific organism to which the disease is due.

*Prognosis.*—During the acute stage, while the disease is still advancing, it is impossible to say how far it will extend; if the intercostal muscles become affected and the diaphragm is alone carrying on respiration, the occurrence of slight broncho-pneumonia is sufficient to cause a fatal issue.

It is, however, most exceptional for a child to die during the acute stage of the disease. It may be stated as a general rule that the amount of paralysis in the acute stage is considerably in excess of that which is present in the later stages, so that a child who at the onset is paralysed in all four extremities will frequently recover completely so far as the arms are concerned, leaving one leg completely paralysed and the other weak, so too when the whole of one leg is at first affected, the residual paralysis may be limited to a single group of muscles.

The more rapid the improvement during the first month after the onset the better the prognosis; on the other hand, some cases improve very slowly, and the improvement may extend over as long a period as two years. After two years very little improvement is to be looked for, but here again exceptions are met with in which the disease has been of several years' standing, and in which by graduated exercises considerable improvement is obtained. It has seemed to be that such improvement has taken place in cases in which artificial supports have been used early, and no attempts have been made to make the best use of the muscles that remain.

In a few cases recovery is complete, no trace of the disease remaining after four to six weeks. In others no trace of weakness can be recognised until the child is undressed, and then slight wasting of the muscles of one thigh or limb is all that can be detected, and all movements are well performed. Such cases are the exception, and recovery always takes place rapidly in these.

It is well to bear in mind the possibility that a progressive muscular atrophy may occur at a later age, but the probability is so remote that the question need hardly be considered in regard to prognosis.

*Diagnosis.*—The diagnosis of the disease is, as a rule, simple; the acute onset, the loss of power, and the flaccidity of the limb without any disturbance of sensation are usually sufficient to make the diagnosis clear.

During the early stages of the disease, owing to the pain which the child has, and the fact that the child resents any movement, the disease is often regarded as rheumatism or some acute affection of the bones or joints.



In acute epiphysitis there is often a sudden loss of power with complete flaccid palsy of one extremity, generally the arm. This affection commonly occurs in infants of a few weeks to a few months old who are the subjects of congenital syphilis, but in many ways this paralysis closely simulates acute anterior poliomyelitis. The following points usually make the diagnosis clear, the child is usually considerably younger than the age at which infantile paralysis occurs, the paralysis is not complete, there is often some thickening about the epiphysis, but this again is not always present when the flaccid condition of the arm is first noticed. The amount of pain on movement of the joint in these cases is often very slight. When the epiphysis at the upper end of the humerus is involved there is complete flaccid palsy of the whole limb, and when that at the lower end there is wrist drop. The electrical reactions are unaltered.

Injury to the brachial plexus may give rise to flaccid palsy. This may occur either at the time of birth or at some subsequent period. The injury that occurs at birth is usually the result of traction on the arm and a history of prolonged labour, and that the paralysis was noticed immediately after delivery is obtained. The injury is not uncommonly confined to one set of muscles, of which the most commonly involved are those supplied by the fifth cervical root, viz., the deltoid, biceps, and supinator longus.

Without a distinct history of the paralysis having existed since birth, the diagnosis is often difficult, for the absence of sensory changes and trophic disturbance of the skin does not exclude the possibility of some peripheral lesion.

*Injury to the Brachial Plexus after Birth.*—Cases are sometimes seen in which there is a definite history of injury followed by paralysis, and this paralysis is attributed to injury of the brachial plexus. In many of the cases the amount of the affection is out of all proportion to the nature of the injury, for instance the whole arm may be paralysed and all the muscles absolutely flaccid, and this may be attributed to a fall on the shoulder. Such a widespread palsy would involve all the roots from the fifth cervical to the first dorsal. Here again the absence of all sensory impairment and the absence of trophic changes in the skin would all point to an affection of the anterior horns rather than to one of the peripheral nerves.

Infantile hemiplegia or monoplegia will sometimes give rise to an appearance which at first sight may somewhat resemble infantile paralysis. The limb may be wasted and cold, there is marked loss of power, and there may be some deformity of the foot. The limb, however, is generally somewhat rigid, the knee-jerk is exaggerated, ankle clonus may be present, though this is not common, and the plantar reflex may be of the extensor type, although in this connection it should be remembered that when the calf muscles are paralysed it is not unusual to obtain extension of the great toe on stimulating the sole of the foot. The affection of the arm and leg on the same side would also be in favour of a cerebral lesion, but it must be remembered that occasionally when the arm is affected by infantile paralysis the leg on the same side is sometimes weak from involvement of the pyramid on the same side. In cases of doubt the question can be easily settled by an electrical examination of the muscles.

Congenital spastic paraplegia is sometimes mistaken for infantile paralysis, though in a typical form of the disease the error seems hardly possible. In some cases, when there is retraction of the flexor muscles, the knee-jerks are not obtainable, so that the two conditions might be mistaken. The spastic condition of the muscles should at once serve to distinguish the disease from infantile paralysis.



Myopathy may occasionally be mistaken for an anterior poliomyelitis; but it is more frequent for a widespread anterior poliomyelitis to be mistaken for a myopathy, more especially when there is affection of the extensor muscles of the thighs and the muscles of the back, for then the position assumed by the patient in rising from the floor is exactly that of a child with myopathy. The gradual onset, the wide distribution of muscles affected, and the grouping of the muscles so affected, generally render the diagnosis easy. An electrical examination of the muscles would also aid in the diagnosis, for in the case of a myopathy there is simple quantitative alteration without any qualitative change.

From a multiple neuritis either following diphtheria or some other infection or cause, the slower onset, the less complete and the more widely distributed paralysis, and the concomitant affection of one or more of the cranial nerves, together with the fact that complete recovery gradually takes place, make the diagnosis easy.

Hæmorrhage into the spinal cord has been diagnosed as infantile paralysis, but the extreme rarity of the affection would hardly ever lead to its suggestion unless some unusual etiological circumstance were present.

*Treatment.*—If the patient is seen during the acute stage of the disease, the pain in the back, which is often considerable, may be relieved by the application of leeches, hot fomentations, and counter-irritation. The child should, if possible, be made to lie in the prone position in bed. If convulsions are present, or the child is very restless, the administration of bromide may considerably relieve these symptoms. The ordinary treatment of the febrile condition which accompanies the disease should be adopted, and purgatives, salicylate of soda, and quinine, may be given. Hyperpyrexia should be treated by tepid sponging.

After the immediate acute stage has passed and the paralysis has become established, the inunction of mercury, the administration of belladonna, ergot, or iodide of potash seem sometimes to have a beneficial effect.

At a later stage strychnine may be given with advantage.

The local treatment of the paralysed limb is at this stage most important, and that which is of the greatest moment is that the limb should be kept warm. As it has already been stated, there is very considerable vaso-motor disturbance, and the limb readily gets cold.

Warmth should be kept up in the limb by friction with the dry hand, and in the intervals the limb should be covered in a loose-fitting knitted stocking extending well above the paralysed part. Artificial warmth may with advantage be applied in the form of dry heat, but it is necessary to adjust the temperature with care, for, owing to the vaso-motor disturbances, burns which may be produced heal very slowly. In order to prevent deformity, it is well to place the leg at night into a light poroplastic or leather splint, so arranged that it shall keep the foot at right angles to the leg.

Massage may with advantage be commenced as soon as the child is free from pain, in order to keep the muscles in as good a condition as possible while recovery is taking place. Passive movements form a most important part of this treatment, and should be carried out more frequently than the actual rubbing. It is well that such movements should be repeated at least three times a day. Resistance movements are also of importance, and even in a child who is unable to make any movement with the paralysed limb, it is well to make the child do the movements with the sound limb, with the object, if possible, of producing some movements in the paralysed limb.



With regard to the application of the electric current much depends on the reaction of the muscles. If the paralysed muscles react to a faradic current of a moderate strength, its application may be beneficial, and can do no harm, except in so far as it tends to unduly develop the normal muscles, and eventually to increase the amount of contraction, owing to the inequality in the strength of the muscles. If the paralysed muscles no longer react to faradic stimulation, but react to galvanism, then it is advisable to use that form of current, the positive or negative pole being applied to the muscle according to which evokes the better response. There is, however, one great difficulty connected with the constant current, and that is that most children strongly object to it; they will often stand a faradic current so strong that it will set the observer's muscles into tetanus, and yet they will not tolerate a galvanic current that will give rise to a contraction. Various methods can be adopted for its application, either direct with one electrode on the back and another on the limb, or by placing the child in a bath and passing the current through the water. Although of service, too much importance should not be attached to the application of electricity, and if the child cannot stand the current sufficiently strong to cause a contraction of the muscle, it had better not be persevered with. The treatment of the later stages of the disease is mostly surgical, and the only question that arises is the time at which an instrument for the support of the limb should be applied. There is no doubt that the power of recovery is prevented by the early application of an instrument. To say that instruments should not be worn for two years after the onset of the disease would be a too dogmatic statement if applied to all cases; but if no deformity is produced, it is well to allow that time to elapse before an instrument for the support of the limb is ordered.

Apart from instrumental support, various operative procedures may have to be resorted to, the division of tendons for the correction of deformity, arthrodesis, the transplantation of tendons, and in severe cases amputation may be advisable. Each case has to be judged by the extent of the paralysis and the deformity produced, and for details of operative procedure the reader is referred to some work on orthopædic surgery.

**ACUTE ANTERIOR POLIOMYELITIS IN THE ADULT.**—Acute anterior poliomyelitis in adults runs so different a course from the disease seen in the infant that it entails a separate description. Not only is the course of the disease different, but its progress and prognosis are by no means so favourable.

*Onset and Course.*—The onset of the disease is usually attended by a rise in temperature. The loss of power does not occur with the suddenness that is seen in the case of infants, but manifests itself more gradually in the course of one or more days. The paralysis may, as in the infantile form, affect one limb, but more commonly it affects both legs and both arms, spreading from the former to the latter. Such a palsy in the infant would probably show a tendency to rapid improvement, but not so with the adult; it remains stationary or only slowly improves.

Atrophy of the affected muscles rapidly occurs, while on electrical examination they show the reaction of degeneration. In advanced stages of the disease they undergo further change, and contracture occurs.

Sensation remains intact; but even the presence of some impairment of sensation does not necessarily exclude the possibility that the condition is an anterior poliomyelitis.

Sometimes there is severe pain in the back and in the limb affected.

The majority of adult patients who are affected are attacked between 20 and 30 years of age.



The prognosis is unfavourable. It is said that complete recovery may take place, but in the majority of cases but little recovery is seen.

With regard to the ætiology of the affection, it seems to follow very much the same affections as are seen in the infantile form—that is to say, after severe cold, and the specific fevers, measles, diphtheria, influenza, and after prolonged exertion.

*Diagnosis.*—The diagnosis of the affection, which is simple in the child, is in the adult a matter of considerable difficulty, since there are other affections in them which closely resemble poliomyelitis, and which occur far more frequently than this affection. The most important of these is a polyneuritis, and it not infrequently happens that in that disease a history is obtained of sudden loss of power. Sensory disturbances should theoretically form a diagnostic feature; but they may be present in anterior poliomyelitis, and may sometimes be absent in a polyneuritis. Tenderness of the muscles and trophic changes in the skin would be in favour of a polyneuritis, while an affection of muscles not corresponding to a spinal segment would point in the same direction. It must, however, be admitted that the distinction between poliomyelitis and polyneuritis is in many cases impossible; in fact, it is probable that the whole lower neurone is affected, and that those which are longest are most prone to suffer.

*Hæmorrhage into the Spinal Cord.*—The rapid onset of paralysis caused by hæmorrhage into the spinal cord may suggest the diagnosis of an acute poliomyelitis, and the flaccid paralysis of the legs and the loss of knee-jerk which often accompany it also point in the same direction. The later stage of the disease is essentially different, and the alteration in sensation and the spastic condition of the legs, with increased knee-jerks, usually enable a correct diagnosis to be made.

**SUBACUTE POLIOMYELITIS.**—The occurrence of this form of poliomyelitis, as apart from progressive muscular atrophy, has been doubted; but the distinctive character of the disease is that paralysis first appears, and is followed by muscular atrophy, which is not the case in the disease known as progressive muscular atrophy.

*Etiology.*—The disease occurs in association with pregnancy and with metallic poisoning. It is also said to occur in individuals who have been the subject of acute poliomyelitis in infancy.

*Symptoms.*—The disease begins gradually with weakness in one or other extremity, followed after a few weeks or months by weakness in another limb. Following on this loss of power there is muscular atrophy, fibrillary tremor, and a partial or complete reaction of degeneration. The malady is progressive, and is fatal either by involving the respiratory centres or by some pulmonary affection.

*Prognosis.*—The disease is usually progressive.

*Diagnosis.*—The diagnosis from a progressive muscular atrophy lies in the fact that the disease is usually more widespread, and that the paralysis appears before the muscular atrophy.

*Pathology.*—The pathological identity of these cases is not established; in some the process would seem to be similar to that found in the acute poliomyelitis of infants, namely, a vascular lesion, while in others a primary atrophy of the cells of the anterior horn has been found.

In the vascular form localised foci with cells infiltration, and destruction of the ganglion cells, and finally a condition of sclerosis are found.

In the second form there is a general atrophy of the cells of the anterior horns and of the ground substance of the gray matter. In many of the cases there is scattered degeneration of the antero-lateral tracts.



CHRONIC SPINAL MUSCULAR ATROPHY AND BULBAR PARALYSIS.—There are two affections, “progressive muscular atrophy” and “chronic nuclear bulbar paralysis,” that usually receive separate descriptions, but which may with advantage be considered together, in that, from the pathological standpoint, they are of the same nature. To these may be added a third affection, “amyotrophic lateral sclerosis,” which is also essentially the same. The underlying morbid condition in these three affections is a progressive degeneration of the motor neurones of the spinal cord, medulla oblongata, and brain respectively. Even clinically these affections present certain features in common; but different types can, nevertheless, be distinguished according as the symptomatology indicates an affection of the motor neurones of the anterior horns of the spinal cord (progressive muscular atrophy); similar affection of the neurones of the motor nerves in the medulla oblongata, the cells of which nuclei are the homologues of the anterior horn cells of the spinal cord (bulbar paralysis); or implication of the motor neurones of the cerebral cortex, in conjunction with those of one or other, or both of the lower levels, in which case sclerosis of the lateral columns of the spinal cord is added, and the affection is known as “amyotrophic lateral sclerosis.”

*Etiology.*—It is exceptional for more than one member of a family to be attacked, and only in very rare instances has there been any evidence of direct transmission from parent to offspring, so that these affections contrast strikingly with the myopathic forms of muscular atrophy in which heredity plays so prominent a part. Adults are alone affected, and most commonly between the ages of 40 and 50; the bulbar form of the disease does not occur in young subjects, and although the spinal form may do so, it is rare for even this form to begin before the age of 25. Men are more often affected than women. In many cases no exciting cause can be determined, but various causes have been assigned in different cases, and it seems possible that some of these may have something to do with the generation of the disease. Of intoxications, syphilis and lead have each been credited with a deleterious influence of this kind. Blows to the back and injuries that cause general shock to the nervous system have been supposed to be effective, and there are cases in which atrophy has commenced in a limb that has received an injury. Exposure to cold, sexual excess, anxiety, mental strain, and worry have all been regarded as exercising an unfavourable influence of the same kind.

*Morbid Anatomy.*—The precise changes met with in the nervous system depend on which neurones are affected, spinal or bulbar, or both, and as to whether or not the neurones of the sensori-motor cortex are concomitantly involved with degeneration resulting in their axones in the pyramidal tracts. It is the exception to meet with cases in which degeneration of the pyramidal tracts is not present, though a few such cases have been recorded. In cases of this kind, although there is no degeneration of the pyramidal tracts, there may be some degeneration of the white matter of the ventral part of the spinal cord in the region of the ground bundles. The essential change in this class of case is a degeneration and atrophy of the cells of the anterior horns. There may be an entire disappearance of the cells from some parts of the cord, while in other parts some cells remain, though many of these are much reduced in size and are obviously atrophic. The fine nerve fibres which course through the anterior horns are also reduced in number, and there is much increase in fibrous tissue, which replaces the degenerated nerve elements. All these changes lead to shrinking of the anterior horns, which are accordingly much reduced in



size, it may be to an unequal degree on the two sides, when the disease is more advanced on one side than on the other. No round cell infiltration, or other evidence of inflammatory reaction, is present, although in some cases the blood-vessels are found engorged.

The degenerative changes are usually most pronounced in the cervical part of the spinal cord, to which region they may be limited; but the lumbar part of the cord is, of course, similarly affected in those cases in which the muscles of the lower extremities have been atrophied during life.

When the motor nuclei of the medulla and pons are implicated there is degeneration and atrophy of the cells of their neurones, in every way similar to the degeneration which occurs in the cells of the anterior horns of the spinal cord. The fine nerve fibres that course through the nuclei degenerate and disappear, and the nerve elements are replaced by proliferation of connective tissue. The hypoglossal nucleus is the chief seat of the degenerative changes in these cases, but similar though usually less pronounced changes are met with in the facial, the motor fifth, and the vago-glossopharyngeal nuclei.

In those cases in which there have been spastic phenomena during life, and even in others in which there has been no suspicion of this, extensive degeneration of the pyramidal tracts is found, and it may be possible to trace the degeneration of the axones that comprise these tracts from the sensori-motor region of the cerebral cortex to the lowest part of the spinal cord. Moreover, the cells of these neurones in the cortex are degenerated and atrophied; many of them are shrunken and their axones and dendrites are atrophied and broken off, while the cells of other neurones have entirely disappeared. It is the large pyramidal cells of the cortex that undergo this atrophic change, and the degeneration of their axones can be traced through the corona radiata, internal capsule, crus, pons, and medulla oblongata to the spinal cord. The degeneration of these axones may be seen in the spinal cord alone, however, without evidence that the degeneration extends all the way up the pyramidal system to the cortex, or it may be traced from the cord up to the medulla or pons, while the motor path above this is free from degeneration.

These changes in the pyramidal system are met with in association with degeneration of the cells of the anterior horns of the spinal cord, or of those of the motor nuclei in the medulla oblongata, and pons, while in other cases both the motor nuclei in the bulb and the anterior horns are affected in conjunction with the degeneration of the pyramidal tracts.

*Pathology.*—The changes met with in the central nervous system are due to a slow progressive degeneration of the motor neurones, whether these be affected in the anterior horns, in the nuclei of the medulla and pons, or in the cerebral cortex, though why there should be this premature death of the nerve elements is a question that has yet to be solved. The possibilities are that there is a congenital predisposition to early decay, or that some toxic agent, capable of selective action, attacks the motor neurones. The most generally accepted view as to the structural independence of the upper and lower neurones makes it clear that, whatever the influence may be which determines the degeneration and death of the nerve elements, the lower neurones of the cord and medulla are affected independently of the upper or cortical neurones, and that there is not a direct extension of the degeneration from the upper to the lower neurones. So, too, in that the cell is the part of the neurone that preserves the nutrition of the whole unit, it is easy to understand why, with gradual decay of the neurone, the



part farthest from the cell should first show signs of death, and that accordingly the axones commence to degenerate at their most distal part. This is the way in which degeneration of the pyramidal tract in the spinal cord and medulla is to be accounted for, when no demonstrable evidences of degeneration are detected in the pyramidal cells of the sensori-motor cortex of the brain.

*Symptoms.*—In that the clinical picture of the disease is different according as the neurones of the anterior horns of the spinal cord, those of the bulbar nuclei, or those of the sensori-motor cortex, are affected, the symptomatology of the three forms deserve separate descriptions.

**PROGRESSIVE MUSCULAR ATROPHY.**—The onset of this affection is, as a rule, without numbness, pain, or other subjective sensations. The patient gradually develops weakness, with muscular atrophy, usually in the upper limbs, and most frequently the small muscles of the hands are first affected. Weakness of the thumb is usually the first defect noticed, movements such as are necessary for picking up a pin or in buttoning the clothes become difficult and eventually impossible, and before long the patient may recognise that the ball of the thumb is flat on one side as compared with the other. If the patient comes under observation at this stage of the illness, atrophy of the thenar eminence and weakness of the movements of the thumb, notably those of abduction and opposition, can be determined. If it has not preceded the atrophy of the thenar muscles, as is most common, there soon follows hollowing of the space between the thumb and index finger, as viewed from the back of the hand. This is due to wasting of the first dorsal interosseous muscle, in consequence of which the index finger cannot be properly abducted from the middle finger. The other interossei also waste, so that the spaces between the metacarpal bones all show a variable degree of hollowing, proportional to the degree of atrophy that has resulted, while movements of abduction and adduction of the fingers are feeble. The hypothenar eminence also becomes flat owing to atrophy of the muscles of this part of the hand, and abduction of the little finger is weak. If the interossei become markedly atrophied before the long extensors and flexors of the fingers are affected the *main en griffe* results, for the interossei, when normal, flex the fingers at the metacarpo-phalangeal joints, and extend them at the phalangeal joints, so that when they are paralysed the long extensors and flexors bring about over-extension at the metacarpo-phalangeal joints and flexion at the phalangeal joints, and the hand thus assumes a claw shape. If the lumbricales be also atrophied the flexor tendons stand out prominently in the palm of the hand, owing to the great hollowing that results.

Although the muscular atrophy begins on one side it always becomes bilateral, so that it is rare for many months to elapse before similar symptoms appear on the side that is not at first affected; moreover, the affection is, as a rule, symmetrical as well as bilateral, identical parts being attacked on the two sides.

As the disease progresses the muscles of the forearm next become atrophied, and the flexors usually succumb before the extensors. The deltoid is next involved, and the shoulder loses its rounded contour and becomes flat, while in advanced cases a depression appears beneath the outer end of the acromion process. The supra- and infra-spinati become atrophied about the same time, and the subscapularis and teres major and minor soon share their fate, as do the biceps, brachialis anticus, and supinator longus. The serratus magnus usually becomes affected, in consequence of which the vertebral border of the scapula projects when the arm



is raised to the horizontal line in front of the patient. The upper or clavicular portion of the pectoralis major as a rule wastes, as do the trapezius and rhomboids; but it is noteworthy that the trapezius is only affected in so far as its middle and lower portions are concerned, the upper part of the muscle practically always escapes,—a fact that led Duchenne to speak of it as the *ultimum moriens*. The levator anguli scapulæ commonly escapes, and the latissimus dorsi, lower half of the pectoralis major, and triceps are almost always unaffected.

The neck muscles may become atrophied and weak, though the sternomastoid usually escapes, and the platysma myoides is never affected. The muscles at the back of the neck, as a rule, suffer more than those in front, so that the head falls forward, and the chin rests against the upper part of the sternum; but the patient can jerk the head unduly backwards, and can then balance it in this position on the spine.

The intercostal muscles commonly suffer in the later stage of the affection, and they may become completely paralysed, in which case respiration is carried on by the diaphragm alone, but, on the other hand, it may be the diaphragm that is chiefly affected, in which case the patient has to depend on the intercostals for the preservation of life. How precarious the patient's existence may be at this stage of the malady may be gathered from the fact that the intercostals rarely escape entirely even when there is a preponderating weakness of the diaphragm. Less commonly the abdominal muscles become atrophied and weak.

The lower extremities may remain unaffected throughout the whole course of the disease; but when affected the muscles that waste are usually the anterior tibial group, the quadriceps extensors of the knee, and the glutei. There are rare cases in which the atrophy begins in the lower extremities before the muscles of the upper limbs are affected, in which case the anterior tibial muscles are the first to suffer.

Throughout the course of this disease the amount of paralysis detected is proportional to the degree of the muscular atrophy that is present. Moreover there is no spasticity such as is due to sclerosis of the lateral columns of the cord, and such as results in the form of the affection that has been named amyotrophic lateral sclerosis. Any contracture that is present is due to the unopposed action of muscles that have escaped atrophy when their antagonists have been affected. The tendon jerks remain unaltered, unless the muscles on which they depend become much atrophied, in which case they are abolished, so that the knee-jerks are preserved, except in the cases in which the extensors of the knee are wasted; but the tendon jerks do not become exaggerated, and ankle clonus is never present.

The plantar reflex is preserved as long as the muscles that produce the movements which constitute this reflex are intact; and as in the normal subject, the toes always flex when the sole of the foot is stimulated. There is never any blunting of cutaneous sensibility. The sphincters escape except in rare instances, and then they usually only suffer late in the course of the disease, though there are exceptions to this rule. Sexual power is not uncommonly lost.

The clinical picture of a case of progressive muscular atrophy may conform to the above description throughout its whole course; but, on the other hand, phenomena due to affection of motor nuclei of cranial nerves may supervene, in which case bulbar paralysis is added to the spinal cord affection. So too, with or without this addition, the clinical picture may be altered to that of amyotrophic lateral sclerosis by the appearance of



spastic phenomena which indicate degeneration of the lateral columns of the spinal cord.

AMYOTROPHIC LATERAL SCLEROSIS.—In this form of the disease the upper and lower motor neurones are both involved, and there is thus evidence of lateral sclerosis in addition to muscular atrophy.

The mode of onset varies. In some cases the earliest manifestation is a spastic condition of the limbs, and the tendon jerks are increased all over the body. Muscular atrophy is added later, and is in every way similar to that already described under progressive muscular atrophy. A second group of cases commence like cases of progressive muscular atrophy, the earliest sign of disease in them being atrophy of muscles, but sooner or later the limbs become stiff and the tendon jerks exaggerated. In a third group the spastic phenomena and the muscular atrophy come on together, and it is not possible to say that the one symptom preceded the other.

It was on cases of the first kind that Charcot based his classical description of amyotrophic lateral sclerosis, and it is probable that the other forms are still imperfectly recognised as being examples of the same disease.

The patients complain of weakness and it may be of stiffness in the limbs, and on physical examination either no muscular atrophy is detected, or such as is present is not sufficient to account for the weakness. As the rigidity in the limbs increases movements at all the joints are hampered by the stiffness, and the gait presents the characters of the spastic type. The knee-jerks are found to be exaggerated, ankle clonus is commonly present, the arm-jerks are all increased, and it may be possible to evoke a marked jaw-jerk. Moreover, stimulation of the sole of the foot may elicit an abnormal plantar reflex, in that the toes may extend instead of flexing as they should under normal circumstances. When muscles begin to atrophy in these cases it is usually the arms that are earliest affected, in which case the first dorsal interosseous muscle or the small muscles of the thumb are first attacked, while the subsequent spread of the muscular atrophy is in the same order and distribution as in progressive muscular atrophy. It may, however, be muscles supplied by cranial nerves that are first attacked, in which case bulbar paralysis results, and the manifestations of the disease are then identical with those to be subsequently described under chronic nuclear bulbar paralysis. Atrophy of the muscles of the lower extremities occurs less commonly, and does not, as a rule, become nearly so pronounced as in the muscles of the upper limbs.

In consequence of spasticity and contracture of the muscles that do not atrophy, the upper arms may be more or less glued to the sides of the thorax, the elbows may be partly flexed, the forearms pronated, and the wrists and fingers flexed. As the atrophy of the muscles increases, however, the spasticity lessens; indeed, the muscular atrophy may be so widespread and so pronounced in the upper limbs that but little muscle is left to allow rigidity to be determined. In the lower extremities the rigidity is always very marked, and as long as progression is possible the gait is pronouncedly spastic. The rigidity may lessen with the appearance of muscular atrophy in the lower limbs, but never to anything like the same extent as in the upper extremities.

The neck muscles, the intercostals, and the abdominal muscles all become affected in the advanced stages of this form of the disease, as in the cases in which there is no evidence of lateral sclerosis. A very large proportion of the patients develop bulbar symptoms sooner or later, so that it is more common to meet with cases of amyotrophic lateral sclerosis in

which bulbar paralysis forms part of the clinical picture than not, and hence it is that these patients are so frequently emotional. So, too, respiratory and cardiac troubles occur, which are probably due to affection of the cells of pneumogastric nucleus.

There is no blunting of sensibility; but the subjects of this affection not uncommonly complain of numbness, tingling, and pain in the limbs. The sphincters if affected at all only become so in the final stages of the disease.

**CHRONIC NUCLEAR BULBAR PARALYSIS** (Glosso-labio-laryngeal Paralysis).—This form of bulbar paralysis is essentially of the same nature as progressive muscular atrophy, the only difference being that the neurones of some of the motor cranial nerves are affected instead of those of the anterior horns of the spinal cord. The disease may begin in this part of the central nerve axis, and may run its course to a fatal termination without any evidence of similar affection of the neurones of the anterior horns of the spinal cord. In other cases the anterior horns become affected, and then the symptoms of progressive muscular atrophy are added to those of bulbar paralysis, in the same way that symptoms of bulbar paralysis may arise in the course of a case that begins as progressive muscular atrophy.

Whether the disease be limited to the neurones of motor cranial nerves, or whether there is affection of the cells of the anterior horns as well, it is common to detect signs of lateral sclerosis in these cases, so that the muscular atrophy is combined with spasticity, and the cases then present the features of bulbar paralysis in association with amyotrophic lateral sclerosis. In short, cases of this kind afford examples of the concomitant affection of the motor neurones of the cerebral cortex, of the nuclei of certain cranial nerves, and, it may be also, of the anterior horns of the spinal cord.

In bulbar paralysis the tongue is as a rule the first part to be affected, and speech becomes defective, notably in regard to the pronunciation of dental and guttural consonants, in that the tongue has to be brought in contact with the teeth in the one case and with the palate in the other. The movements of the tongue are impaired, and the organ shows signs of atrophy. It cannot be protruded to the full extent, nor can its tip be properly curled up, and lateral movements are also impaired, so that it cannot be brought in contact with the cheek on either side. All that is evident in the way of atrophy at first is that the organ is somewhat flattened, but as the atrophy progresses the mucous membrane becomes thrown into longitudinal folds, with grooves between, and the organ then has a shrivelled, puckered appearance. A further feature which is very characteristic is the occurrence of fibrillary tremors in a longitudinal direction in the course of the muscle fibres of the tongue.

Weakness of the orbicularis oris next becomes manifest, so that the patient can neither pout nor whistle, and speech, already defective in consequence of the paralysis of the tongue, becomes further impaired, the chief new difficulty experienced being in the pronunciation of the explosive labials "b" and "p," which become "m" and "v."

Bilateral paralysis of the soft palate accompanies the paralysis of the tongue and orbicularis oris, so that the patient's speech has a nasal quality, and "k" and "g," already difficult of pronunciation, become more so, in that the difficulty of pressing the tongue against the soft palate is increased. The paralysis of the palate also increases the difficulty of pronouncing the explosive labials "b" and "p," in that some of the air escapes by the posterior nares. The escape of air in this way also makes it impossible for



the patient to blow out the cheeks, even if the lips are tightly closed between the fingers and thumb, and the effort to do so merely results in a snorting noise, due to the rushing of air through the nose. When attempts are made to swallow liquids, they regurgitate through the nose, as the palate no longer shuts off the posterior nares from the pharynx during the act of deglutition. On phonation or deep inspiration the palate either does not move at all, or is very imperfectly drawn up, and the reflex action of the palate is also diminished or abolished, so that the uvula is not elevated when the palate is pricked.

Later in the course of the disease the vocal cords may become paralysed; but this does not occur as frequently as the name "labio-glosso-laryngeal palsy" might lead one to suppose. When the vocal cords are affected the voice becomes low-pitched and monotonous, and the patient cannot cough properly. Still later the nucleus of the vagus may become affected, in consequence of which the heart's action becomes rapid, and the patient is liable to attacks of dyspnoea.

Another late phenomenon that may occur is atrophy of the chin muscles, but with the exception of these muscles and the orbicularis oris, all the other muscles of the face, as a rule, escape. Sometimes the muscles of mastication are affected, in consequence of which the lower jaw may drop, the mouth remains open, and the patient can only bite very feebly.

The unfortunate victim of this malady is usually very emotional, and cries and laughs without cause, although the intellect remains perfectly clear. When the disease is fully established the expression is most lugubrious, the mouth remains open, the lips are everted, and the saliva is constantly dribbling from the mouth, where it accumulates in excessive amount owing to the difficulty there is in swallowing it. The power of speech may be entirely lost, so that a few grunts may be all that is possible, or the patient may be able to phonate a few vowels, or mumble a word or two very slowly in a monotonous way. The tongue lies on the floor of the mouth, shrivelled up and incapable of the slightest movement, and the soft palate is pendulous and immobile, or it flaps to and fro with each act of respiration, while thick viscid saliva may cling to the fauces in festoons.

By the time this stage is reached there is commonly little or no power of mastication, and the act of swallowing is exceedingly difficult and attended with considerable danger, in that the larynx cannot be drawn up under the epiglottis, whose muscles are also weak, as are the constrictors of the pharynx; thus food readily passes into the larynx and produces choking, and in that there is little power of effective cough, suffocation or inhalation pneumonia may be induced.

Reflex action of the palate is abolished or is very feeble, so that when the palate is touched it either does not move at all or is only feebly raised, and the pharyngeal reflex is also abolished, so that tickling the fauces fails to induce retching.

The tendon jerks are not increased in the cases of bulbar paralysis unaccompanied by lateral sclerosis, nor in the cases in which, without affection of the lateral columns of the cord, the neurones of the anterior horns of the spinal cord are degenerated in conjunction with those of the nuclei in the medulla oblongata. When the bulbar paralysis forms part of the clinical picture of the form of the affection in which there is lateral sclerosis, and which is known as "amyotrophic lateral sclerosis," then it is that the tendon jerks are increased. In such cases a jaw-jerk, or even jaw clonus may be present, the arm-jerks are increased, as are the knee-jerks, and



ankle clonus can be elicited. So, too, the toes are extended instead of being flexed when the plantar reflex is elicited.

*Diagnosis.*—The conditions from which chronic spinal muscular atrophy and bulbar paralysis has to be distinguished differ somewhat according to the form that the disease assumes.

*Myopathy.*—When the indications suggest progressive muscular atrophy it is of primary importance to exclude the muscular atrophies of myopathic origin, and attention to the following points usually makes this task comparatively easy:—The absence of a hereditary history of the disease, the more advanced age at which the manifestations appear, the presence of bulbar symptoms, fibrillary tremors in the muscles, and evidence of the reaction of degeneration on electrical stimulation, all support the diagnosis of the spinal cord affection. The combination in which the muscles are atrophied is, moreover, different in the two varieties of disease, for while the small muscles of the hands commonly suffer in the spinal cord affection, they usually escape in the myopathies, and although in both forms of disease the muscles about the shoulder girdle may be attacked, it is noteworthy that the deltoid frequently suffers in the former class while it usually escapes atrophy in the myopathies. So, too, the trapezius may atrophy throughout its whole extent in the latter class of affection, but its upper portion escapes to the very end, as a rule, in the spinal cord disease. Then, again, atrophy of the lower half of the pectoralis major and latissimus dorsi is highly characteristic of one form of myopathy; whereas these muscles are rarely affected when the atrophy is due to changes in the spinal cord. The detection of enlargement of any muscles negatives the possibility that the muscular atrophy is of spinal origin.

In addition to the points of distinction that have already been noted, the presence of spasticity and exaggeration of the tendon jerks serve to distinguish amyotrophic lateral sclerosis, in that these phenomena never occur in the myopathies. Moreover, although the face is affected in the facio-scapulo-humeral form of idiopathic muscular atrophy, the tongue, palate, and larynx never become involved, so that bulbar paralysis is readily distinguished from this form of myopathy.

*Subacute Poliomyelitis.*—The onset is more rapid, paralysis as a rule precedes atrophy of the muscles, there is usually some tendency to recovery, and the muscles that are atrophied are picked out at random, and not in the definite order that obtains in progressive muscular atrophy.

There is another form of muscular atrophy that is usually subacute in onset, in which some muscles of the hands and arms are picked out, while others escape in a manner that suggests affection of the spinal cord, but whose pathology is as yet uncertain. These cases are further distinguished from progressive muscular atrophy owing to the fact that pain is commonly an early symptom, and it may be possible to detect some anæsthesia.

*Acute Anterior Poliomyelitis* differs from the chronic affection of the anterior horns in the fact that the onset is sudden and attended with rise of temperature. The paralysis that is at first present exceeds that which subsequently persists, the muscular atrophy follows on the paralysis, and only some of the muscles that are at first disabled become wasted, and the reaction of degeneration is more pronounced in them than when the muscular atrophy is due to the chronic degenerative disease. Moreover, children are usually attacked, whereas it is extremely rare for the chronic form of affection to commence in childhood.

*The peroneal type of muscular atrophy*, in which there is degeneration in the peripheral nerves and posterior columns of the spinal cord, is distin-



guished by the following features :—Although the small muscles of the hands waste, the atrophy commences in the peronei and other muscles of the leg below the knee, the onset of the disease is in childhood, several members of the family are similarly affected, and there may even be a hereditary history of the affection. Further important points of distinction from progressive muscular atrophy are the occurrence of pain, blunting of cutaneous sensibility, and vasomotor changes in the skin.

*Peripheral neuritis* usually causes pain or tingling sensations, the nerves are tender to pressure, and some anæsthesia can usually be determined. Moreover, the onset is more rapid in neuritis, paralysis precedes muscular atrophy, and corresponds to the anatomical distribution of the nerve affected; moreover, the reaction of degeneration is more complete. Fibrillary tremors do not occur, and all spastic phenomena are absent.

When the neuritis affects the cervical nerve roots the diagnosis is usually more difficult, for the same muscles may then be affected as in progressive muscular atrophy, and there may be very little blunting of sensibility; but the paralysis is of more rapid onset, and pain is usually a prominent symptom.

*Cervical pachymeningitis* may also be difficult to distinguish, owing to the similarity in the distribution of the muscular atrophy, and from the further fact that spastic phenomena may be present in the lower extremities, suggesting amyotrophic lateral sclerosis. There is, however, a good deal of rigidity about the neck, without any similar condition in the arms, pain is usually severe, there may be tenderness on pressure over the cervical spines, and some anæsthesia can as a rule be determined. It is, further, important to remember that bulbar symptoms never occur in these cases.

*Caries or tumour* at the level of the seventh cervical and first thoracic vertebræ may occasion atrophy of the small muscles of the hands, and spastic phenomena in the lower limbs. But pain in the back, pain radiating along the nerve roots, tenderness on pressure over the spine at this level, the occurrence of angular curvature, the absence of spasticity in the arms, the presence of anæsthesia, and the frequency of sphincter affection, all serve to distinguish these cases from amyotrophic lateral sclerosis.

*Syringomyelia*.—The distribution of the muscular atrophy in this disease, affecting as it usually does the small muscles of the hands, and the presence of spasticity and exaggerated tendon jerks in the lower extremities, may suggest amyotrophic lateral sclerosis, but the absence of spasticity in the arms, the presence of anæsthesia, the occurrence of painless whitlows and other trophic disturbances of the skin, nystagmus, and lateral curvature of the spine, all serve to make the diagnosis clear.

*Primary Spastic Paraplegia*.—Although the spastic phenomena are the same as in amyotrophic lateral sclerosis, there is no atrophy of the muscles such as occurs in the latter disease.

*Disseminated Sclerosis*.—In this disease also, although it is common to meet with spastic phenomena, muscular atrophy is rare. Moreover, optic nerve atrophy, nystagmus, the peculiar speech defect, intention tremor, and early affection of the sphincter of the bladder, make the diagnosis of the disseminated disease certain.

*Myelitis* is of more rapid onset, sensation is commonly affected, as are the sphincters, while it is rare to meet with atrophy of the small muscles of the hands in this affection.

The differential diagnosis of chronic nuclear bulbar paralysis will be dealt with when all the forms of bulbar paralysis have been described (see p. 157).

*Prognosis.*—Though always grave, the prognosis varies with the form that the disease assumes. It is best in progressive muscular atrophy without indications of lateral sclerosis, and most unfavourable in bulbar paralysis, while amyotrophic lateral sclerosis stands intermediate between the other two forms as regards the seriousness of the outlook. In progressive muscular atrophy there may be arrest of the disease, either spontaneously or as a result of treatment, and the arrest may be permanent; but in other cases the disease subsequently progresses to a fatal termination.

Muscles that no longer respond to electrical stimuli cannot be expected to recover, but when the damage to the muscles has only been slight it is possible that there may not only be arrest of the atrophy, but even some improvement in their nutrition. The gravity of the case is always much increased when there are indications of bulbar paralysis, or when the muscles of respiration are involved; but as long as the muscles of the limbs are alone affected there are no grounds for apprehension as to the immediate future. A fatal issue is inevitable in bulbar paralysis, and the end is usually reached within two years from the onset of the affection. The deterioration is as a rule steadily progressive, though in some cases the affection remains stationary for a time, only to progress again, however, after this temporary lull. Death may result from paralysis of respiration, owing to extension of the morbid process to the nucleus of the vagus, or the patient may die from asthenia consequent on the ingestion of an insufficient amount of food. Life may, on the other hand, be cut short by suffocation owing to impaction of food in the glottis, or by bronchitis or pneumonia induced by inhalation of liquids and food particles into the lungs.

*Treatment.*—Every effort must be made to improve the patient's general nutrition in the hope that the morbid tendency of the nerve elements to degenerate may in this way be lessened. A liberal dietary, supplemented by cod-liver oil and extract of malt, fulfil these requirements in so far as progressive muscular atrophy and amyotrophic lateral sclerosis are concerned; but when there is bulbar paralysis the feeding of the patients becomes a matter of serious moment. A careful watch has to be kept over them while they are at meals, for food may readily become lodged in the glottis and cause fatal suffocation. They may experience difficulty in swallowing both liquid and solid foods, though happily they can as a rule swallow soft pultaceous foods better; but even this becomes impossible in the final stages of the disease. Life has then to be preserved by artificial feeding by the nasal tube.

Fresh air, especially in the country or by the sea, is beneficial, and even a sea voyage may be expedient in some cases. All depressing influences, whether physical or mental, must, as far as possible, be avoided, hence, although exercise is indicated, it must never be taken in such amount as to cause fatigue.

The most careful precautions are needed to prevent these patients from taking cold, for in many of them the muscles of respiration are weak, and they are thus ill-prepared to combat any bronchial or pulmonary affection. They ought always to be warmly clad, and should wear flannel next to the skin.

All tonic medicines are of service in the treatment of these affections, so that arsenic, iron, quinine, and phosphorus are all indicated. Strychnia is of special service in the treatment of progressive muscular atrophy and bulbar paralysis in which there are no signs of lateral sclerosis; but it is contra-indicated in amyotrophic lateral sclerosis, for it tends to increase the



spasticity that is present in this form of the disease. The drug is supposed to be more effective when administered by subcutaneous injection than when given by the mouth (Sir Wm. Gowers).

Local treatment by means of galvanism and massage is most important for improving the nutrition of the affected muscles. In making use of the galvanic current the positive pole ought to be placed at the back of the neck, and the negative pole should be stroked over the muscles, while the strength of current used ought to be just enough to evoke a distinct contraction of the muscle. The current should be applied to the tongue and palate as well as to the facial muscles when there is bulbar paralysis. Massage not only improves the nutrition of the muscles, but may possibly reduce the amount of spasm in the amyotrophic lateral sclerosis form of the disease, while passive movements are of service in preventing or reducing contracture.

**BULBAR PARALYSIS.**—*Definition.*—An affection in which the lesion is in the medulla oblongata and pons, or in which it involves the cranial nerves derived from these parts, so that paralysis of the lips, tongue, palate, pharynx, and larynx results, and in consequence there is articulatory disturbance of speech, which is also nasal in quality, difficulty in swallowing, and it may be aphonia.

Cases of bulbar paralysis fall into one of two classes, acute or chronic, according to the mode of onset of the symptoms, and the former class includes a variety in which the onset is sudden, and another in which it is rapid, though not sudden. Moreover, the manifestations of the different forms of the affections differ according as the lesion is supra-nuclear, nuclear, or infra-nuclear in position.

There are also two affections known respectively as “pseudo-bulbar paralysis” (double hemiplegia), and “asthenic bulbar paralysis (myasthenia gravis) that may be considered with advantage in this connection.

**ACUTE BULBAR PARALYSIS.**—The sudden form of acute paralysis is always due to a vascular lesion, most commonly thrombosis or embolism, though hæmorrhage accounts for some cases of the kind. Most of the people that are attacked are at or beyond the middle period of life, which is accounted for by the frequency of atheroma then, but young people may suffer owing to embolism, or as a result of thrombosis consequent on syphilitic arteritis.

The onset is quite sudden: a patient may go to bed feeling perfectly well, and may wake next morning and find himself paralysed, or the attack may come on during the day, in which case giddiness is commonly experienced, and the patient may vomit. Consciousness is lost in some cases. The paralysis that results is usually bilateral, but commonly affects structures on the two sides unequally and irregularly; it is exceptional to meet with strictly unilateral paralysis, though cases of the kind do occur. The lips, tongue, soft palate, pharynx, and larynx may all be involved. The palate hangs motionless during attempts at phonation, the tongue can neither be protruded nor moved from side to side in the mouth, and the lips cannot be pursed as in the attempt to whistle. Articulation may be impossible and phonation weak, while deglutition is difficult, and food either regurgitates through the nose or enters the larynx and induces coughing.

Instead of complete paralysis of such wide range the palsy may be less severe, and some parts may be affected while others escape. Thus, with complete paralysis of the tongue only one side of the palate may be affected, and the larynx may escape, or the palate and pharynx may be affected while the tongue and larynx escape. On the other hand, even with palsy of



partial distribution the lesion may be more widespread so as to occasion paralysis in the distribution of other cranial nerves, as, for instance, the facial, the motor or sensory division of the fifth, or both. Moreover, the effects of the lesion may be still more widely felt, in that there may be weakness in the limbs, usually hemiplegic in distribution, or blunting of sensibility on one side of the body, and in the absence of objective signs there may, nevertheless, be subjective sensations such as numbness or tingling in these parts. Such widespread manifestations are especially liable to be met with at the outset, whereas the symptoms may subsequently clear up wholly or partially, leaving only the bulbar paralysis, which also commonly improves to some extent, though a variable degree of defect of this kind is permanent. Indeed, when seen in after years the patient's condition may closely resemble that met with in chronic bulbar paralysis due to progressive degeneration of the nuclei of the bulb; but unlike that affection there is no tendency for the paralysis to progress.

There is an acute inflammatory form of bulbar paralysis in which the onset though rapid is not sudden; several hours, or even a few days, may elapse before the maximum effect of the lesion is produced. The morbid process that attacks the bulbar nuclei under these circumstances is the same as that met with in acute poliomyelitis, in which the gray matter of the anterior horns of the spinal cord is the seat of the lesion. The changes consist in destruction of the cells of the nuclei, swelling and disintegration of the nerve fibres, dilatation of the blood-vessels, which are engorged, and exudation of leucocytes, or even actual small extravasations of blood.

The attack is ushered in by febrile disturbance, and with the rise of temperature malaise and headache are experienced, while vomiting occurs in some cases, and there may be diarrhoea. The precise distribution of the paralysis varies in different cases. There may be difficulty in swallowing, paralysis of the palate, tongue, and commonly of the face also, notably its lower half. Like poliomyelitis which affects the spinal cord, parts may be picked out while others escape, so that the face and tongue may be only affected on one side, although both sides of the palate are paralysed and deglutition is difficult.

**CHRONIC BULBAR PARALYSIS.**—This variety of bulbar paralysis is due to either nuclear or infra-nuclear lesions. The nuclear form has already been described in conjunction with progressive muscular atrophy and amyotrophic lateral sclerosis; but we have yet to deal with the chronic forms of infra-nuclear bulbar paralysis, including the effects of lesions of the bulbar nerves outside the cranial cavity.

*Infra-Nuclear Bulbar Paralysis.*—The grouping of the symptoms differs according to whether the nerves are implicated in the intra-medullary part of their course, or whether they are affected outside the medulla oblongata before they leave the cranial cavity, and there is a further difference in the symptomatology when the nerves are involved outside the cranial cavity.

*Intra-medullary lesions* are rare, but may occasion a form of crossed paralysis in which half of the tongue and soft palate and one vocal cord are all paralysed on the same side, while the limbs are affected on the opposite side, and the face escapes. Hemianæsthesia may also be present on the side that the limbs are paralysed. The affected part of the tongue wastes, and the muscles of this side of the organ, together with those of the corresponding half of the palate, show the reaction of degeneration on electrical examination.

*Extra-medullary lesions* occur more frequently, and are most often due to syphilitic affections, either gumma or chronic meningitis; but simple



meningitis may also lead to the symptoms that characterise this form of bulbar paralysis, as may tumours that are situated near the medulla. The symptoms that arise are paralysis of half of the tongue and palate, together with similar affection of the vocal cord on the same side; but what is distinctive of a lesion in this situation is the association of paralysis of the sterno-mastoid and upper part of the trapezius with the bulbar paralysis, owing to implication of the spinal accessory nerve. The muscles and the affected half of the tongue waste, and present the reaction of degeneration on electrical stimulation. As the structures on one side are alone affected, no bulbar symptoms arise, for as long as the muscles of the opposite side are intact, no difficulty in speaking or in swallowing is experienced.

*Extra-cranial affections*, such as caries, tumours, cellulitis, or peripheral neuritis may cause paralysis in the distribution of the lower bulbar nerves by affecting them after they have left the cranial cavity, and it is easy to mistake cases of this kind for those due to intra-cranial lesions; indeed, the differential diagnosis may be most difficult. Caries or new growth, by involving the pharyngeal plexus of nerves, may lead to paralysis of the palate without affection of the tongue or larynx, while neuritis may cause weakness of the tongue, palate, and vocal cords, or only a single nerve may be affected, as, for instance, one hypoglossal, with paralysis and atrophy of the corresponding half of the tongue.

**PSEUDO-BULBAR PARALYSIS (Double Hemiplegia).**—In that the muscles supplied by bulbar nerves are represented in both cerebral hemispheres, a lesion in one hemisphere cannot cause paralysis of these muscles, and, accordingly, bulbar symptoms do not result from unilateral cerebral lesions. When there is a lesion in both cerebral hemispheres, however, and double hemiplegia results, bulbar symptoms are present, and the condition has been designated “pseudo-bulbar paralysis.” In these cases there is always a history of two attacks of hemiplegia, first on one side and then on the other. With the first attack no bulbar symptoms arise other than the ordinary articulatory difficulty of speech commonly present in hemiplegia; but if a second attack causes paralysis of the other half of the body bulbar symptoms result. There may be inability to raise the upper lip so as to show the teeth, the tongue is not properly protruded, speech is impossible, or is characterised by a marked nasal quality, or the patient may even be unable to phonate. Swallowing is difficult, and the soft palate may not move at all, or but little, on attempts at phonation. Despite these defects the muscles do not atrophy: they respond normally on electrical excitation, and reflex action is preserved in the soft palate and pharynx. The limbs are weak on both sides, though like the face the side more recently affected may be weaker than that first attacked (see also “Hemiplegia,” vol. iv.).

*Asthenic bulbar paralysis* (Myasthenia gravis) is described elsewhere (see vol. viii.).

**Diagnosis.**—The problems to be discussed in the diagnosis of bulbar paralysis differ according to whether the acute or chronic form of the affection is under consideration.

The first point to be decided in a case of acute bulbar paralysis is whether the symptoms are due to a lesion in the pons or medulla, or whether the case is one of pseudo-bulbar paralysis consequent on a lesion in each cerebral hemisphere. In the latter class of case there is always a history of an attack of hemiplegia at some time before the attack in which the bulbar symptoms arise; the reflex irritability of the palate and pharynx is preserved, the muscles do not waste, and they respond normally on electrical excitation.



If it is decided that the lesion is in the bulb, it next becomes a question as to whether it is supra-nuclear, nuclear, or infra-nuclear in position. In such considerations we receive valuable help from the state of nutrition of the affected muscles, their mode of response on electrical stimulation, and the state of reflex excitability of the palate and pharynx. If the lesion is situated in the motor path above the nuclei of the bulbar nerves, then the muscles do not waste; they continue to respond normally on electrical stimulation, and the reflex action of the palate and pharynx are preserved; but if the nuclei, or the nerves in their intra-medullary course after they have emerged from their nuclei, are involved the affected muscles waste, as may be determined in the tongue and lips. Moreover, the atrophied muscles present the reaction of degeneration. The alterations in the electrical excitability of the muscles can be determined in the palate as well as in the tongue; and, moreover, the reflex excitability of the palate and pharynx are either diminished or abolished.

It is usually difficult to discriminate between cases in which the defects are due to affection of the bulbar nerves as opposed to implication of their nuclei in that the cases have so many points in common. We, however, derive some help from the fact that lesions which involve some of the nuclei of cranial nerves occasion paralysis of more muscles than are supplied by the nerve itself. Thus a lesion that involves the hypoglossal nuclei may cause paralysis of the orbicularis oris as well as of the tongue, whereas when the nerve is affected the tongue is alone paralysed. So too the proximity of the hypoglossal nuclei to each other makes it unlikely that one would be involved in a lesion without the other, except in poliomyelitis. On the other hand, the hypoglossal nerve is sufficiently near to the pyramidal tract to make some implication of the motor path possible when a lesion involves this nerve in its intra-medullary course, hence evidence of crossed hemiplegia is in favour of a lesion of the nerve as opposed to affection of its nucleus.

The points that serve to distinguish the chronic nuclear form from asthenic bulbar paralysis (myasthenia gravis) are referred to in connection with the latter affection (see "*Myasthenia Gravis*," vol. viii.)

Cases in which an acute lesion has caused widespread defects in the distribution of the bulbar nerves are distinguished from chronic nuclear bulbar paralysis by the history of acute onset and by the non-progressive character of the affection.

The same facts that help us to decide between nuclear and infra-nuclear affections when the lesion is acute hold good when the chronic forms of bulbar paralysis are under consideration. Extra-medullary lesions are almost always unilateral, and are further characterised by paralysis of the trapezius and sterno-mastoid, in conjunction with similar affection of the half of the tongue, palate, and larynx on the same side.

*Prognosis.*—In acute bulbar paralysis the chief danger is that the patient may die when the manifestations are still acute; but many of them survive this stage, and prognosis then becomes much better than in the chronic forms of bulbar paralysis, for even complete recovery is possible, though rare, while some improvement results in all patients that do not succumb during the acute stage. Most improvement may be expected when the lesion is supra-nuclear, and least when the nucleus is involved. When cases that are due to vascular lesions are under consideration, it must be remembered that both thrombosis and embolism may recur, and that although recovery may result after one attack a fatal issue may be determined by some future attack.



The chronic degenerative form of bulbar paralysis always terminates in death, and that usually within two years from the onset of symptoms. Extra-medullary lesions are less serious than intra-medullary, and owing to the possible beneficial influences of treatment syphilitic affections are the least serious. Tumours in this situation are usually rapidly fatal.

*Treatment.*—In cases of sudden onset, when there is reason to suppose that thrombosis consequent on syphilitic arteritis is the cause of the bulbar symptoms, energetic treatment by mercury and iodide of potassium ought to be adopted. Apart from this the principal things to be attended to in acute cases are the feeding of the patients and the treatment of the paralysis that remains after the acute stage of the illness is over. When the patient is unable to swallow he must be fed by the nasal tube. The paralysis calls for no special treatment when the lesion is supra-nuclear; but when it is nuclear or infra-nuclear the muscles atrophy, therefore the constant current ought to be applied daily to all the affected parts. Of internal remedies strychnia, iron, and quinine are most useful, and the strychnia may be given with advantage by subcutaneous injection, when there are indications of respiratory or cardiac failure during the acute stage of the disease.

The points to be attended to in the treatment of cases of chronic progressive bulbar paralysis have already been indicated (see p. 153). In chronic cases in which the lesion is infra-nuclear, antisiphilitic treatment by mercury and iodide of potassium has to be relied on, in the hope that the condition may prove to be syphilitic. Similarly in children cod-liver oil, malt, iron, and other tonics are administered owing to the possibility that a tumour may be tuberculous. The only other treatment that is possible is surgical intervention in extra-medullary tumours.

#### IV. PARALYSIS WITH TREMOR OR ATAXY

**DISSEMINATED SCLEROSIS.**—*Definition.*—A disease in which areas of sclerosis are scattered irregularly throughout the nervous system, and in which the most characteristic symptoms are a combination of spastic and ataxic paraplegia, loss of control over the sphincters, notably that of the bladder, weakness with intention tremor in the arms, nystagmus, optic nerve atrophy, and scanning speech.

*Etiology.*—Hereditary transmission of the disease has only been very exceptionally observed. More often there is an indirect neuropathic inheritance in the form of some other nervous disease that has attacked some of the patient's ancestors. Young adults are most commonly affected between the ages of twenty and thirty years, and it is quite exceptional for the disease to commence later than forty. Cases have been described in children, but there seems little doubt that the majority, if not all that were recorded by earlier writers, were not genuine. Owing to the protracted course of the malady few of the suspected cases came to autopsy in childhood, and those that did, and that were supposed to establish the occurrence of the disease in children, are now discredited. Competent observers have, however, since recorded cases of the affection in children, and in some instances it has been possible to trace symptoms of the affection met with in adults back to childhood for their commencement. One sex is as liable to be attacked as is the other, though in the statistics of different observers one or the other preponderates. There has been a strong tendency among modern writers on the subject to regard the disease as due to the action of toxic agents on the nervous system, and Oppenheim considers that certain



occupations where people are impregnated with metallic poisons, notably lead, are responsible for some cases of the disease. Alcohol has been credited with a similar influence. But in that it is quite impossible to discover any evidence of such intoxications in the large majority of people attacked, some more subtle influence probably underlies the pathology, even in those cases where toxic agents are accessory factors in causation.

The infective fevers have been supposed to supply the intoxication, and prominent among those suspected of this have been enteric fever, influenza, and malaria; but it is obviously difficult to disprove that these febrile illnesses have not merely lighted into activity a morbid process that had its inception at some antecedent period. There seems little doubt that the cases supposed to be the outcome of malaria have not been genuine, but that they have merely presented a group of nervous symptoms, consequent on the intoxication, which have resembled the manifestations of disseminated sclerosis. Rheumatism and pneumonia have each preceded the appearance of the disease in a few cases, and some observers have attempted to establish an etiological relationship between syphilis and disseminated sclerosis, but there seems to be little justification for this.

The antecedent disease may be some affection of the nervous system, most often some form of myelitis. Blows and injuries have been regarded as causes, as have exposure to cold and chills, indeed some observers have considered the latter influences as the most common causes of the malady. It is, however, obviously difficult to exclude the possibility that trauma or cold only serve to stimulate into activity some pre-existing though dormant morbid condition. The same may be said of fatigue, which probably only serves to intensify phenomena that really existed, though they had not been previously noticed. The same relationship exists between disseminated sclerosis and emotion; moreover, any of these influences are capable of causing relapses, or may accelerate the progress of the disease. So, too, the first manifestations of the malady have been observed during pregnancy, and it is certain that pregnancy and parturition exercise an unfavourable influence on the course of disseminated sclerosis.

*Morbid Anatomy.*—Patches of sclerosis are scattered at random throughout the nervous system, any part of which is liable to be attacked, though there appears to be a predilection for certain regions. The areas of sclerosis are usually found both in the brain and spinal cord, though in a few exceptional cases that have been recorded it is possible that the disease was really limited to the brain or cord, and that islets of sclerosis were not overlooked in the part supposed to be unaffected. The peripheral nerves would appear to be much less liable to be attacked; but they have not received the same amount of careful examination that has been bestowed on the central nervous system, so that it is possible that future investigations may prove that the peripheral nerves do not enjoy the immunity that has been supposed.

Although the whole nervous system is liable to be attacked there are certain seats of election; thus, in the cerebral hemispheres the white matter of the centrum ovale, and the gray matter of the basal ganglia, are much more often invaded than is the gray matter of the cortex. In the spinal cord the patches of sclerosis are most plentiful in the white matter, and invasion of the gray matter is less frequently seen, though it does occur, and an island of sclerosis may be even limited to the gray matter in some part of the cord. The cerebellum is, as a rule, less affected than other parts of the central nervous system, and, as in the case of the cerebrum, the white matter is more often attacked than the gray cortex. No parts are respected



in the crus, pons, and medulla oblongata, where the sclerotic areas are scattered at random through the gray and white matter.

We possess very little definite information with regard to the peripheral nervous system, except in so far as certain of the cranial nerves and spinal nerve roots are concerned. Sclerosis may invade the nucleus of a nerve or its fibres in their intra-medullary course, or after they have left the central nervous system. Any of the cranial nerves may be thus affected, though the optic and olfactory nerves are most commonly attacked. They have, however, been found intact when other cranial nerves have been invaded. In the case of the optic nerves the patches of sclerosis may occur in some part of the nerve itself, or in the tract or chiasma.

Both motor and sensory spinal nerve roots may show changes, and the nerves of the cauda equina have been found degenerated, so that it is possible that more systematic examination of the peripheral nerves will reveal changes in them also.

As seen with the unaided eye, before the brain and spinal cord have been placed in a preservative fluid, the islands of sclerosis are of a warm gray colour, or they are reddish gray or brown, and they have a peculiar translucent appearance; indeed, they may be quite gelatinous looking. They vary in size from quite minute islets up to patches two or more centimetres in diameter, and they appear to be sharply demarcated from the surrounding healthy tissues. These areas of sclerosis may be seen on the external surface of the part affected, or they may be only revealed on section.

Microscopical examination may confirm the impression received on macroscopic examination, that the transition from normal to abnormal structure is abrupt, but in other cases the transition is seen to be gradual. The former state of things is especially liable to be seen at the lateral limits of an area of sclerosis which affects a tract of parallel fibres, whereas the ends of such a patch may show a gradual transition from healthy to morbid structure.

One of the most extraordinary features of the disease is the remarkable power of resistance shown by the axis cylinders, for even when the myelin sheaths of the nerve fibres are completely destroyed, the axis cylinders, as a rule, remain intact. This escape of the axis cylinders has been supposed to explain the still more remarkable fact that secondary degeneration of the nerve element does not occur, except very rarely, and then limited, as a rule, to a very short distance in the immediate neighbourhood of the patch of sclerosis which causes it. Thus sclerotic tissue may interrupt the various long tracts in the spinal cord, and other parts of the central nervous system, without any ascending or descending degeneration resulting.

The cell bodies of the neurones also escape until late in the course of the disease, as a rule, and much sclerosis may exist in their immediate neighbourhood without the cells themselves showing any evidence of structural change. The escape of this part of the peripheral motor neurones, in the anterior horns of the spinal cord, explains why muscular atrophy and the reaction of degeneration are so rarely met with in disseminated sclerosis.

There is marked proliferation of the interstitial tissue, and large numbers of glia cells are, as a rule, seen; the dense mass of fibrils are arranged longitudinally when they occur in nerve tracts of parallel fibres, but they form a feltwork of interlacing fibrils when seen in the gray matter, and the central part of such a mass is dense, while a loose meshwork is seen at the periphery where the connective-tissue fibrils radiate in all directions, and insinuate themselves between the nerve elements. In the earlier stages of



the morbid process there is marked cell proliferation, but little evidence of fibrillary structure, while, when the disease is more advanced, the glia cells are more separated, and the meshwork of fibres becomes more evident. The oldest areas may appear homogeneous, and, in addition to the elements already described, they may contain large spider cells whose processes form a meshwork the interstices of which are homogeneous in appearance.

The blood-vessels remain normal in some cases, while in others their walls are thickened, and the perivascular spaces may be dilated. The vessels are said to be narrowed, and even thrombosed in some cases. The capillaries may be increased in numbers, and they may be engorged, so that white blood cells escape into the surrounding tissues, and even small hæmorrhages may occur.

Such slight thickenings or adhesions of the pia mater as may occur are clearly secondary to affection of the nerve elements.

*Pathology.*—There is little that is certain and much that is debatable with regard to the pathology of disseminated sclerosis. We are not yet in a position to decide how the disease originates, nor are we able to say whether the morbid process is primarily interstitial, or whether the nerve elements succumb first. The former is the view that is most widely accepted, and some observers indeed regard the disease as a form of chronic myelitis; others, however, hold that a parenchymatous degeneration of the nerve elements occurs first, and that the hyperplasia of the interstitial tissue is secondary to this, a view that accords well with the growing belief that the morbid changes are induced by the action of a toxic agent. Moreover, it has been suggested that a proliferation of interstitial tissue, initiated in this way, may, by its extension, lead to secondary destruction of the nerve elements outside the original starting-point of the morbid process in each affected area. Another hypothesis is that the morbid changes are secondary to affection of the blood-vessels; but it seems more probable that the altered state of the vessels and the morbid changes in the nerve elements are due to a common cause. Yet another suggestion that has been made is that the morbid process is a multiple gliosis, which depends on congenital influences, but that although essentially endogenous, the impetus to disturbance may be due to some exogenous cause such as an acute disease or a trauma.

*Symptoms.*—Although the disease is essentially cerebro-spinal, and there is little to warrant the distinction of a cerebral as opposed to a spinal form, yet in its clinical manifestations symptoms due to affection of one part of the central nerve axis may dominate the clinical picture; when this is so it is much more common to find that the symptoms indicate an affection of the spinal cord and not the brain, for, as a rule, when cerebral symptoms are pronounced, there are also indications of affection of the spinal cord.

The mode of onset and subsequent course of the illness present great variations, but in the large majority of cases the most constant symptom of the earlier stages of the established disease is paraplegia, in which the weakness is associated with spasticity, and it may be also ataxy. Many cases begin in this way, and the only diagnosis warranted by the physical signs at this stage is "primary lateral sclerosis" where spasticity exists alone, and "ataxic paraplegia" when ataxy is added. In either case, however, other phenomena subsequently appear which establish the diagnosis of disseminated sclerosis.

In a very large proportion of cases, although some form of paraplegia is commonly present when the patient first comes under observation, there is a history of some antecedent manifestation of the disease, of which there



may still be tangible evidence, or which may have entirely disappeared. Indeed, these symptoms are so fleeting that they are commonly regarded as "hysterical," more especially when women are attacked in this way, and when physical or mental shock has immediately preceded the appearance of the symptoms. Loss of sight in one eye, diplopia, and aphonia are some of the evanescent symptoms that have been noticed. So, too, some part may be affected by numbness, or some other subjective sensation such as "coldness," "deadness," or "pins and needles." Probably the most common of the evanescent symptoms is, however, loss of motor power in one or more limbs, while in exceptional cases convulsions occur.

The great feature of this type of the disease is that these early symptoms may entirely pass away, but they return in the same or different form, and with similar abruptness, at some subsequent period; or, without wholly clearing up, they may lessen and may remain in abeyance for months or years. Even where recovery is supposed to have taken place, close interrogation will often elicit that sight has been a little defective at times; that diplopia has occasionally been noticed; that the patient has limped when tired, or that from time to time she has been a little hysterical in manner. These are sure indications that there is but a temporary lull, and that the disease will certainly burst into activity again at some future period, near or remote, when the same symptoms may return in more pronounced form, or new symptoms may arise. Fresh activity of the morbid process may occur without obvious exciting cause in some cases; while in others it can be clearly traced to some debilitating influence such as pregnancy and parturition, some form of acute illness, privation, exposure to cold, traumatism, or mental shock. Even when the symptoms are in the main progressive, there is a remarkable tendency to periodicity, transient improvements being followed by renewed activity of the disease. Many such remissions may occur before death, and, although this event may be delayed for a great many years, it is the only way in which this disease terminates.

The classical symptoms of the established disease are spastic paraplegia with or without ataxy added, exaggerated tendon jerks, some defect of the sphincter of the bladder, intention tremor, which may be more widespread, but which is usually most pronounced in the upper limbs; nystagmus, optic nerve atrophy, scanning utterance and psychical disturbances, notably of an emotional character. It must not, however, be supposed that all of these symptoms are present in every case of the disease, or that the absence of any of them necessarily militates against the diagnosis. It would, for example, be as erroneous to suppose that the diagnosis of disseminated sclerosis is not warranted in the absence of nystagmus, as to imagine that the diagnosis of tabes is not justified if the pupil reaction to light is preserved. Nevertheless, most if not all of the symptoms that have just been narrated, as a rule, appear before the disease has completed its course.

So varied are the manifestations of this ubiquitous disease that it will be of greater advantage to consider the individual symptoms in detail than to attempt to sketch a clinical picture that will be suited to all cases.

*Psychical State.*—The patients are commonly emotional, and laugh immoderately, or cry without adequate cause. Later in the course of the disease their manner may suggest a state of wellbeing that is not justified by the physical condition, while still later they may become fatuous, and may ultimately sink into a state of dementia.

*Speech.*—True aphasic defects are very rare, but speech is commonly altered in a characteristic manner that has been variously described as "scanning," "syllabic," or "staccato." Words are spoken slowly, and with



pauses between the different syllables, each of which is unduly accentuated; but the end of the word may be slurred over, and words may be unduly separated from each other. The disease may, however, reach a stage when other symptoms make the diagnosis clear, and yet speech may not be affected. The voice is commonly monotonous, and it may be tremulous, or it may be nasal in quality owing to paralysis of the palate, or various degrees of aphonia may be met with. The first words of a sentence may be spoken better than those towards the end of it, as the muscles concerned with speech and respiration in common with other muscles in this disease are easily fatigued.

*Ocular Phenomena.*—Defects of vision are common, and these may or may not be attended with changes that can be detected in the optic nerves on ophthalmoscopic examination. Loss of sight may be one of the earliest symptoms, and may be rapid or even sudden in onset, while in its subsequent course remissions may occur in the way that is so characteristic of many symptoms in this disease. Sight is usually only defective in one eye at first, and vision may remain good in the other eye for years. But in such long-standing cases changes can be seen in both discs on ophthalmoscopic examination, though they are only slight on the side on which vision is not affected. The optic discs become pale, one eye is affected before the other, and the temporal half of the disc is most commonly attacked when the change is partial. The appearances are due to simple atrophy of the optic nerves, which is the usual condition met with; but some observers have described slight optic neuritis in a few cases. There may be amblyopic defects of vision, and even concentric limitation of the field, and yet the ophthalmoscope may reveal no change in the optic nerve, a state of things probably due to retrobulbar affection of the nerve. On the other hand the disc may be pale, and yet the acuity of vision may be normal; indeed such pallor may precede any defect of vision for many years. Central scotoma for colours, and concentric limitation of the visual fields are met with in a considerable proportion of cases.

Nystagmus is the most common affection of the ocular muscles, though it is not present in a considerable proportion of cases; it is most frequently horizontal, but may be vertical, rotatory, or of mixed form. The nystagmus may be spontaneous, but more often it is only evoked on voluntary movement of the eyes, in which case it is more commonly evoked by lateral movements than when the patient looks up or down. Both eyes are usually affected, though the nystagmus may be dissimilar in the two; and in rare instances it affects one eye alone; it may only appear when the eyes are turned to one side, or it may then be more pronounced than when they are turned in the opposite direction.

Paralysis of ocular muscles occurs in a much smaller percentage of cases, and though defect of this kind may be present when the patient comes under observation, in many cases there is only a history of it, as diplopia or squint once present has disappeared. The palsies are commonly of central origin, and conjugate turning of the eyes to one side and convergence are most often defective. Of isolated affections of the ocular nerves paralysis of the sixth is the most frequent, though the third may be similarly affected, and the fourth occasionally suffers alone. The pupils are usually equal and react normally; but they may be unequal, and, exceptionally, they do not respond to light or on accommodation, the latter being the more common defect.

*Motor Disorders.*—In addition to the disturbances of speech and the defects of the ocular muscles, motor disorders occur in the trunk and limbs.



The most characteristic of these is a peculiar jerky tremor that appears when a voluntary movement is made, and which ceases when the muscles are brought to rest again. This intention tremor consists in a to-and-fro jerking, which is slowly rhythmical, and which increases in range towards the completion of some voluntary act. It is most often seen in the arms, and may be so violent that a glass of water cannot be raised to the lips without its being spilled; so too the patient may be unable to control the tremor sufficiently to be able to touch the point of the nose, or some other object. In slight cases the tremor is only observed towards the completion of some such act, and it is much smaller in range. Similar unsteadiness may be seen in the neck muscles, so that the head shakes as long as it is supported on the shoulders, but all tremor ceases immediately the head is allowed to rest on a pillow. Tremor of the trunk may be observed when the patient is sitting or standing, and tremor may be so pronounced in the lower extremities that standing becomes impossible. The facial muscles may be similarly affected, the nystagmus is of this character, and even the vocal cords may present the same defect, which may be evidenced by a tremulous character of the voice, and which may be seen on laryngoscopic examination.

Motor paralysis is a constant feature of the disease; but the degree and distribution of the motor weakness varies greatly in different cases; moreover, remissions and exacerbations occur in the same patient, more especially in the earlier stage of the malady. It is not common for severe paralysis to result; all movements can usually be performed, though in a slow and feeble way, and fatigue is as a rule readily induced. The lower extremities are most often affected, but the arms are almost as frequently involved. In the former the weakness is combined with spasticity, and in the latter with intention tremor—a combination that is also present in the neck, and which probably accounts for the speech defect in disseminated sclerosis. Palsies also occur in the distribution of cranial nerves, so that in addition to affection of the ocular muscles, those supplied by the facial nerve, the muscles of mastication, tongue, palate, and vocal cords may all suffer.

*Gait.*—There may be inability to stand or walk, but when progression is possible the gait is most often paretic, and there is some spasticity and ataxy as well; either of the added factors may be the prominent feature, so that the gait may be mainly spastic, or markedly ataxic. When there is ataxy it may be of the form seen in tabes, in which the feet are raised too high and the heels are stamped to the ground, or the inco-ordination may be more of the character of that seen in cerebellar disease. The two limbs may be affected differently, or only one may show any defect at first, and rapid variations may occur in the gait as in other symptoms of the disease.

*Muscular Rigidity.*—The muscles commonly become spastic, especially those of the lower limbs, though the muscles of the upper extremities also show some evidence of this. Extensor spasm preponderates in the lower extremities at first, but later in the course of the disease the flexors gain the ascendancy, so that the heels may be pressed against the nates, and limbs become fixed in this position in a state of contracture.

*Tendon Jerks.*—The tendon jerks are as a rule increased, both in the upper and lower limbs, and it may even be possible to evoke an active jaw-jerk. Ankle clonus is commonly present.

*Sensory defects* are usually present in some form. A feeling of giddiness is common, and may be more or less persistent, or it may only occur in paroxysms; the quieter the patient can remain, the greater the freedom from giddiness, as movement of any kind aggravates the condition. Head-



ache and pain in the back may be troublesome symptoms, and cramp-like pains may occur in the limbs, and cause the patient much suffering; but lancinating pains, girdle sensations, and gastric crises are rare. Subjective sensations of numbness and the like are common in the parts presided over by both cranial and spinal nerves, though the hands and feet are the parts most frequently affected in this way. Although a subjective feeling of numbness may be pronounced it may not be possible to detect any objective blunting of sensibility in the part; but when discovered, anæsthesia is usually most marked in the hands and feet. Some anæsthesia is more commonly present than was at one time supposed, but it is subject to such great variations that it is not surprising that it often escapes detection. Although permanent anæsthesia may occur, a part that shows distinct blunting of sensibility at one time may appear to be quite æsthetic on another occasion. There may be blunting of sensibility to all forms of impressions, or there may be more difficulty in appreciating one form of stimulus than another. Hemianæsthesia may be present, and may be the direct outcome of the disease, or it may be an added hysterical phenomenon.

There may be considerable disturbance of the muscular sense, either alone or in conjunction with blunting of cutaneous sensibility, so that Romberg's sign may be well marked when the patient stands with the feet together and the eyes closed.

*Superficial Reflexes.*—The most important change that occurs in connection with the superficial reflexes is that on stimulation of the soles of the feet the toes extend instead of flexing as they do in the normal individual. This phenomenon, which indicates affection of the pyramidal fibres, is commonly seen in disseminated sclerosis. Some observers attach importance to absence of the abdominal reflexes as a diagnostic sign, as it is said that these reflexes cannot be obtained in a large proportion of cases of disseminated sclerosis.

*Sphincters.*—Slight loss of control over the sphincter of the bladder is commonly an early symptom, and may only amount to some precipitancy of micturition or hesitation at the commencement of the act; but in other cases the defect is more marked; and there may also be a little weakness of the anal sphincter. Severe affection of the sphincters, leading to complete loss of control over the bladder and rectum, does not usually occur until late in the course of the disease.

*Sexual Disturbances.*—There may be some loss of sexual power early in the course of the affection, but in other cases there is increased excitability.

*Vaso-motor and trophic disturbances,* though not frequent, are more common than is generally supposed. Some cedema of the feet and ankles may appear early, and, though most noticeable when the limbs are dependent, it may persist in some degree when the limbs are in a horizontal position. The phenomena that constitute erythromelalgia are present in some cases, in others bleb-like blisters appear on the skin, whilst in others swelling of joints has been observed, notably the small joints of the hands and feet. The surface temperature may be lowered in some part, as for instance a limb, and there may be sweating of the part. Bedsores belong to the final stages of the disease, when absolute loss of control over the sphincters is common, and contributes to their production. The hair is said to fall out in some cases, and trophic changes may occur in the nails. Atrophy of muscles has been observed in a few cases, but this is rare.

Palpitation, dyspnœa, and the appearance of sugar in the urine have been attributed to the occurrence of patches of sclerosis in the medulla oblongata.



*Diagnosis.*—There is no difficulty in arriving at a correct diagnosis in a case of disseminated sclerosis, in which the manifestations conform to the classical account of the affection; but very great difficulty is commonly experienced in recognising the aberrant cases which form so large a proportion of the total number of cases of the disease.

*Hysteria.*—No organic disease of the nervous system is so liable to be mistaken for hysteria as is disseminated sclerosis in its early stage, and in no organic affection is the structural basis so commonly obscured by added hysterical phenomena, so that the detection of hysterical stigmata does not exclude the possibility of an organic substratum. Both affections occur most frequently in young adults, the onset of both may be intimately related to some mental shock or physical injury, which may be of an insignificant character. Intention tremor has been described in hysteria, as has nystagmus, though there ought to be little likelihood of mistaking for true nystagmus the jerky unsteadiness of the eyes which occurs in hysteria, and which is commonly associated with blinking movements of the lids. In attempts to differentiate the two affections, the value of the ophthalmoscope cannot be overrated, for the detection of structural changes in the optic nerves places the organic nature of the disease beyond doubt. True nystagmus and affection of the sphincters are also valuable indications of the organic character of the affection, as are ankle clonus, and extensor response as the plantar reflex.

*Primary Spastic Paraplegia.*—In that some cases of disseminated sclerosis present spastic paraplegia as their earliest and only manifestation, for a time, it is obviously impossible to decide at this stage whether symptoms of the more widespread disease will supervene later. The subsequent course of the case can alone reveal its real nature. In watching for the development of other symptoms, care must be exercised not to mistake for the intention tremor and nystagmus of disseminated sclerosis a slight unsteadiness in the upper limbs, and a little nystagmoid jerking which may supervene, in association with increased activity of the arm-jerks, in cases of spastic paraplegia, presumably due to myelitis limited to the caudal half of the spinal cord.

*Ataxic Paraplegia.*—Much of what has been said with regard to spastic paraplegia applies to this affection also, for many cases of disseminated sclerosis come under observation at a stage when the only manifestations present are those which characterise ataxic paraplegia; the subsequent course of the case can therefore alone definitely determine the diagnosis. The large majority of such cases ultimately prove to be disseminated sclerosis.

*Subacute combined degeneration of the spinal cord* in its earlier stages is not infrequently mistaken for the paraplegic form of disseminated sclerosis in its early stage; but combined degeneration of the cord is much the more probable when the affection is symmetrical, and all four limbs show signs of spasticity and ataxy. The patients are usually older than those most commonly affected by disseminated sclerosis, functional manifestations are absent, there is not, as a rule, any tendency to remission of symptoms, although slight nystagmoid jerkings may occur, pronounced nystagmus is never seen, and the sphincters rarely suffer so early as in disseminated sclerosis, although they become completely paralysed sooner. Anæmia is frequently a prominent symptom, as is irregular pyrexia.

*Hereditary Ataxy.*—The cases of disseminated sclerosis that have been described in children have to be distinguished from Friedreich's ataxy. The occurrence of the disease in more than one member of a family, absence



of the knee-jerks, and the presence of lateral curvature of the spine and pes cavus serve to distinguish these cases from disseminated sclerosis. It may be more difficult to discriminate between cerebellar heredo-ataxy and disseminated sclerosis in that this form of ataxy does not become manifest until between the ages of 20 and 30. The affection of more than one member of a family also serves as an aid in the diagnosis of these cases.

*Tabes.*—When motor weakness is pronounced and ataxy slight, a little care may be needed to distinguish a case of tabes. Failure of the pupil to respond to light is an important point in diagnosis, as is absence of the knee-jerks, in that these defects are so rare in disseminated sclerosis. The patients are usually older, there is commonly a history of lightning pains, there is more frequently difficulty in passing water rather than loss of control over the sphincter of the bladder, and blunting of sensibility is more pronounced and persistent.

*General paralysis of the insane* is distinguished by the fact that the patients are as a rule older. Moreover, there is commonly an antecedent history of syphilis, dementia is more pronounced, and is often preceded by mental exaltation, whereas dementia only occurs late in the course of disseminated sclerosis, and beyond a manner suggesting a state of wellbeing that is not in keeping with the patient's physical condition, there is no exaltation.

*Cerebro-spinal syphilis* may give rise to manifestations that resemble those produced by disseminated sclerosis, but nystagmus, intention tremor, and scanning speech are wanting, while sensory symptoms, including those which suggest that the meninges are involved, are much more prominent, and paralysis of cranial nerves, hemiplegia, and dementia are all more common. In some cases, however, the doubt may be only dispelled by the results of antisyphilitic treatment.

*Pseudo-sclerosis.*—Cases of this kind have not been met with frequently enough to make them a serious obstacle in the diagnosis of disseminated sclerosis. The clinical pictures of the two affections are, however, closely similar; but no structural lesions have been found in cases of pseudo-sclerosis adequate to explain the clinical symptoms (see p. 169).

*Cerebral Diplegia.*—Jerky inco-ordination of the limbs, nystagmus, and spinal defect may make some of these cases resemble disseminated sclerosis; but the fact that symptoms date from birth, or from early childhood, serve to suggest their real nature, and the courses of the two diseases are so different that it is rarely necessary to extend the observations over any great length of time before coming to a conclusion.

*Intra-cranial Tumours.*—In cases in which the tumour is situated in the cerebellum, crus, or pons, the inco-ordination of movement that results may be mistaken for that produced by disseminated sclerosis, while a patch of sclerosis in the cerebellum or in one of its peduncles may give rise to symptoms simulating tumour in these situations. Pressure signs, such as headache, vomiting, and optic neuritis are rarely wanting in cases of tumour, whereas such symptoms are not prominent in disseminated sclerosis, and although optic neuritis is said to occur, in some cases, a condition of choked disc is never seen.

*Paralysis Agitans.*—Patients affected by this disease are usually much older, the tremor is constant during repose, and may cease on voluntary movement, it is always regular, even if extensive in range. The high-pitched piping voice, the mask-like face, and the fixed attitude all combine to make the diagnosis simple.

*Toxic tremors* only bear a superficial resemblance to the tremor of dis-



seminated sclerosis, and in addition to the fact that they are present during repose the other symptoms of disseminated sclerosis are wanting. It is conceivable that cases of chronic alcoholism with much tremor and nystagmoid twitchings of the eyeballs may give rise to the suspicion of disseminated sclerosis; but the tremor is fine and rapid, and affects the tongue as well as the hands; it is lessened by the administration of stimulants and aggravated when they are withheld. The subjects of mercurial tremor are usually in an advanced stage of cachexia which is unmistakable.

*Apoplexy.*—The apoplectiform attacks that occur in disseminated sclerosis are distinguished from those of true apoplexy by the absence of premonitory symptoms and of initial fall of temperature. Moreover, their effects are always transitory, and they are not followed by signs of descending degeneration.

*Prognosis.*—A fatal issue is inevitable; but nothing is more difficult than to foretell, with any decree of accuracy, the probable duration of life. The long periods during which the disease may appear to be stationary, or when actual improvement may occur, are largely responsible for this uncertainty in prognosis. When the disease manifests itself by spinal symptoms only, life is, as a rule, preserved much longer than when cerebral and bulbar symptoms appear early; indeed, the occurrence of bulbar symptoms may be taken to indicate that the duration of life will be short. Cases that show a tendency to remission of symptoms live longer than those in which the disease appears to be steadily progressive; but after the manifestations of disseminated sclerosis become so pronounced that the typical clinical picture is seen, life is not, as a rule, prolonged beyond two or three years, though there are many notable exceptions to this rule. The degree of care that can be bestowed on the patient naturally influences prognosis, especially in the cases in which there is a tendency to trophic disturbance in the skin, retention of urine requiring the use of the catheter, or loss of control over the sphincters. There is great risk of cystitis and pyonephrosis, when there is severe affection of the sphincter of the bladder, so that when present this is a bad element in prognosis.

*Treatment.*—No drug has any power of arresting or retarding the progress of the disease, though silver, in the form of the nitrate or chloride, and arsenic, deserve a thorough trial in any case of the kind. The subcutaneous mode of administering these remedies has been recommended, and it is well to follow this plan in the case of the silver salts; but there seems little advantage in administering the arsenic otherwise than by the stomach. Massage, hydropathic and electrical treatment, deserve careful trial, though the results are no more encouraging than those that attend the treatment of the disease by drugs. Massage and passive movements improve the nutrition of the muscles and lessen the tendency to permanent contracture; but it is necessary to employ these measures with caution, so as not to do harm by inducing undue fatigue. Skilled medical care and good nursing can do much that indirectly influences the course of the malady in a favourable manner, for there are many conditions that aggravate the disease and hasten its unfavourable progress. Most patients suffering from disseminated sclerosis bear cold badly, so that in addition to warm clothing residence in a warm climate during the winter is desirable when practicable. Rest is essential, and the nutrition of the patient must be maintained at as high a standard as possible by the administration of nutritious food, malt, cod-liver oil, and tonic medicines, while daily open-air carriage or wheel-chair exercise is no less important. Depressing emotions, mental or physical fatigue, injury, exposure to wet and cold, and



excesses of all kinds must be studiously avoided, in that they exercise an unfavourable influence on the course of the disease. Female patients must especially be warned against pregnancy. Our knowledge of its etiology is not sufficiently precise to determine what prophylactic measures, if any, are likely to prevent the disease. When there is reason to suspect that some slight and transitory affection of the nervous system is to be ascribed to commencing disseminated sclerosis, patients must be warned to keep up their nutrition and to avoid everything that is known to be prejudicial. A sufficiently close relationship appears to exist between the disease and the infective fevers to make it important that patients should be encouraged to rest and otherwise recuperate for some time after one of these illnesses, notably when there has been evidence of derangement of the nervous system during or following on the attack. Measures for the prevention of intoxication by metallic poisons in the various trades are also important.

**PSEUDO-SCLEROSIS.**—A few cases have been recorded in which the symptoms during life resembled those of disseminated sclerosis, but on necropsy corresponding changes have not been found.

**Etiology.**—Nothing definite is known as to the cause of the affection; but the slight abnormalities that have been detected post-mortem suggest that hereditary syphilis may have some influence in the production of the disease. It has also been supposed that alcohol and other poisons may have a similar influence; indeed, in one case, the illness commenced soon after an attack of enteric fever. The affection occurs in young people, and in one case it commenced as early as the age of twelve.

**Morbid Anatomy.**—A firm, leathery consistence of the brain, especially in the occipital region, has been found in some of the cases; but microscopical examination has not revealed any increase of interstitial tissue in the affected parts, and, moreover, the nerve fibres have been intact. There has been a very slight amount of degeneration in the crossed pyramidal tracts in the upper cervical region of the spinal cord, and in one case this could even be traced to the lumbar enlargement, while in another there was slight degeneration in the area of Gowers's tract, on both sides, in the cervical part of the cord.

**Pathology.**—The morbid changes that have been discovered are not adequate to account for the clinical symptoms, and even these slight changes have not been present in all of the cases. The pathology of the affection is obscure, and, but for the possible influence of syphilis and other poisons, nothing is known of its mode of origin. A suggestion that the clinical phenomena are due to hysteria has been rejected by competent authorities.

**Symptoms.**—The intellect may be weak, speech is usually somewhat scanning, and it may be nasal, or even unintelligible. The expression may be somewhat fixed, the movements of the eyes slow, and diplopia has been a symptom. Tremor may be seen in the tongue, and about the mouth and lower jaw, the head may shake, as do the arms on voluntary movement; but the unsteadiness is not quite the same as the intention tremor of disseminated sclerosis. The limbs are weak and all movements are performed slowly. The lower extremities are stiff, paradoxical contractions may occur, and the tendon jerks are increased. There is very little, if any, blunting of cutaneous sensibility, and the sphincters are intact. The course of the disease is protracted, so that it may last as long as ten years; but during this period there are remissions and relapses from time to time. Moreover, apoplectiform attacks with transient symptoms of hemiplegia may occur.



PARALYSIS AGITANS.—*Definition.*—A disease characterised by tremor, associated with rigidity and muscular weakness.

*Etiology.*—The malady occurs in the second half of life, it is rare before forty, and does not frequently occur after sixty; the decade in which it most often makes its appearance is the fifth, but cases have been described in the second and even the first decade of life.

The male sex is more prone to the affection than the female.

Violent emotion would seem not infrequently to be an exciting cause of the disease, numerous cases are recorded as definitely following fright or some profound mental disturbance; while excitement and cold seem also to be antecedents. Many cases are attributed to injury, and the tremor may begin in the limb injured, and gradually spread to the other extremities.

Some authors consider that heredity plays an important part in the disease, but the evidence on this is not very strong; this is, however, a point on which it is necessarily difficult to obtain evidence, as the patients already in advanced age themselves know or remember little with regard to their parents or grandparents; nevertheless, cases have been recorded in which members of the same family have been affected. The disease may follow on acute specific fevers; typhoid, dysentery, influenza, and malaria have all been blamed, but it is worthy of note that paralysis agitans is more common in England than in malarious countries. Syphilis plays no part in the etiology of this disease.

The occurrence of a tremor indistinguishable from that of paralysis agitans sometimes follows on an ordinary hemiplegia, and this tremor, like that of paralysis agitans, tends rather to cease than to increase on the attempt to perform any movement, and the general appearance of these patients is often singularly like that of patients suffering from paralysis agitans.

*Onset and Course.*—The onset of the paralysis is as a rule gradual, sometimes preceded for a considerable period by pains in the limbs and joints, and cramp in the feet and legs, or in others by a sense of extreme fatigue. In certain cases, however, the onset has been sudden and has followed on some fright or injury. When the onset is sudden it may at first only affect one limb, and may for a long time remain limited to that limb.

Almost all observers are agreed that the disease usually commences in the hands, and that as it advances it affects first the leg on the same side, then passes to the arm on the opposite side, and finally to the leg of that side. There are, of course, many exceptions to this rule, and cases have been recorded in which the arm of one side and the leg of the opposite side were first affected. The head is sometimes involved, and even the masseters, the tongue, and, still more rarely, some muscles of the face may be affected by tremor.

*Symptoms.—The Attitude.*—The attitude assumed by the patient is one of the most characteristic features of the disease. He stands with the back bent, the head forward, and the chin down, the legs are flexed at the knees and hips, and the thighs are adducted and rotated inwards, so that when he walks the knees knock together; the arms are flexed at the elbow and slightly abducted from the side, the hands are flexed on the wrists and hang down over the upper portion of the thighs. The fingers are flexed at the metacarpo-phalangeal joints, and the phalanges are extended, the thumb being opposed to the first or second finger. The impression which is produced by the attitude is that of a person who is about to start forward and yet uncertain when to start. The muscles of the face are rigid and have been described as giving the appearance of quiet majesty and dignity, but the



fixed expression gives as a rule rather an appearance of stupidity. The patient is sheltered from all emotions, his smile is hardly visible, and owing to the lower portion of the face being as a rule more fixed than the upper, the forehead and eyes alone play any part in the expression of emotion.

Although the attitude above described is that which is most common, yet there are cases in which the limbs are held in extension, and the head is retracted instead of being bent forward.

Whatever attitude is assumed, it would seem to be due to a special state of rigidity of the muscles; this at first appears as cramp, followed by stiffness, which is transitory, later the condition becomes more constant and exaggerated at times into true paroxysms, and, finally, the stiffness becomes more and more permanent, and the position of the patient resulting therefrom is that above described.

The most frequent position of the hand is that which has been already described, and has been compared to that assumed by a patient about to write, the fingers being extended and the thumb approximated to the first finger.

The fingers are as a whole, however, flexed towards the palm of the hand and deflected to the ulnar side, a position very similar to that seen in chronic rheumatism, except that there is neither swelling nor lipping of the bones, nor is there any grating on movement.

In the lower limbs the thighs are adducted and flexed at the hip, the knees are flexed, the feet extended and in a position of equino-varus.

In late cases the rigidity may be so marked as to resemble a case of paralysis with contracture; there is, however, no true contraction, but a kind of fixation of the joints produced by the long-continued rigidity of the muscles.

Rigidity is, as a rule, only pronounced in the advanced stages of the disease, occasionally, however, it precedes the tremor.

The limitation and slowness of movement is in part due to this rigidity, but there are two other factors that contribute to this condition: the first of these is the slowness of the cerebral processes, a prolonged interval elapsing between the thought and the act, and between the wish and the realisation—the act is accomplished, but only slowly and with fatigue; the second factor is the muscular weakness.

*Gait.*—Patients commonly present a very characteristic alteration in their gait, there is a tendency either to run forward (propulsion) or to run backwards (retropulsion), or both may be present in the same patient; the former may be brought out by standing the patient up and gently pressing on the back, the latter by lightly pulling on the garment of a patient from behind.

The patients rise slowly and with difficulty from their seat, they hesitate before beginning to walk, but when once started on their forward course they run rapidly forward with short, shuffling steps, and are often completely unable to stop themselves, so that unless they come against a wall or some object of support they fall down. They frequently have considerable difficulty in turning round, and in order to avoid this they adopt various manœuvres; one of these patients, in order to get out of the out-patient room, makes a circle round the room and a rapid exit through the door which is held open for him.

In a patient in whom retropulsion is present the same short, shuffling steps are made in a backward instead of a forward direction, and in many cases the patient is unable to stop himself, and unless supported would fall. Sometimes the phenomenon occurs spontaneously. Although retropulsion



is most commonly seen in paralysis agitans, it is not pathognomonic of the disease, for it has also been described in progressive muscular atrophy.

*Tremor.*—Tremor is usually the first manifestation of the disease; it begins commonly in the fingers and hands, and is at first slight, and occurs only at intervals, gradually increasing both in extent and frequency till it becomes almost constant. Commencing in the hand, it may for a long time remain limited to the one limb; gradually, however, it spreads, involving first the fore arm, then the upper arm, then the leg on the same side, then the opposite arm, and finally the opposite leg. As has already been said, the tremor does not commonly affect the head, but that it does do so in some cases, and that even the masseters, tongue, and face may be involved. Affection of the eyelids has been noted, but the ocular muscles always escape. Tremor of the palate, epiglottis, and vocal cords has been described, but it is very rare for these parts to be affected. The tremor of the fingers is a rhythmical movement, having an almost constant amplitude, and is due to the alternating contraction of the opposing muscles; this movement has been compared to that performed in rolling a cigarette or pencil between the fingers. The movements of the elbow have a lesser amplitude than those of the hand, and those of the shoulder are less than those at the elbow. The handwriting is altered by the tremor, but at first the defects are scarcely noticeable by the unaided eye, though the irregularities can be easily seen by the aid of a magnifying lens. As the disease becomes more pronounced the letters become more irregular, the lines zigzaggy, and the writing small and cramped.

The tremor of the legs is far less obvious than that of the arms, but occasionally it is so marked that while sitting the foot shakes against the floor.

The rate of the tremor is comparatively slow, being between four and five oscillations per second, and as it increases in range so it lessens in frequency. It is present during rest, and tends to cease during the execution of voluntary movement for a few seconds, but this can only be said to be true of the less advanced cases, for when of long standing the tremor persists even on voluntary movement. Gerhardt asserts that the tremor is increased in one-half the cases by attempted movement, and may occasionally be brought out by voluntary movement. During sleep the tremor usually ceases.

The voice has a peculiar monotonous intonation, and there is often a delay in starting, but when once started the utterance is rapid, just as there is hesitation before beginning to walk, so there is hesitation before beginning to speak. There is difficulty in modulating the voice, which may even become whining in character.

Deglutition always remains good, but the saliva often dribbles out of the mouth owing to the rigidity of the muscles of the lower portion of the face.

*Alteration in the Skin.*—Changes in the skin are almost constant in advanced cases of the disease, and consist in thickening and loss of elasticity. The thickening may be so marked that it is almost impossible to raise a fold, and even when a fold is formed it is very slow in returning to its former position again. This change in the skin is attributed to thickening of the subcutaneous tissue.

*Subjective Sensations.*—Patients complain of various subjective sensations, the most frequent being the sensation of heat, so that many of them cannot even bear the weight of the bedclothes. Exacerbations of these sensations of heat frequently occur. They also complain of rheumatic and neuralgic



pains in various parts of the body, and often desire to have changed or to change their position in bed.

If the temperature is examined during one of these heat attacks, no elevation of the central temperature is found, but the peripheral temperature may be raised 1 to 2 degrees. It has been suggested that the constant muscular contractions give rise to this increase of peripheral temperature.

There is no impairment of any form of sensation.

The organs of sense are nearly always intact, though some alteration in vision has been described, such as transient amblyopia and contraction of the fields of vision. The movements of the eye remain unaffected, and are moved quickly, but the head is moved slowly, so that if a patient is asked to look in a certain direction he turns the eyes, but not the head. The sphincters are unaffected, and there are no trophic disturbances of the skin. The muscles do not atrophy, nor do they show any electrical change. The skin reflexes are normal, the knee-jerks are not exaggerated in spite of the rigidity of the limbs, there is no ankle clonus, and the plantar reflex gives a flexor response.

The urine is normal, although it is stated that there is an excess of phosphates passed. The mental faculties of the patients remain good, although they are liable to attacks of depression, and may become suicidal, and towards the end of the disease they may suffer from delusions and delirium. During the latter stages of the disease the patients waste and become extremely thin, they lie absolutely helpless in bed, and bedsores may form over the sacrum. They succumb either to general asthenia or to some intercurrent disease such as pneumonia.

*Pathological Anatomy.*—Many of the pathological conditions described by older writers cannot be regarded as of value, since they did not distinguish between such diseases as disseminated sclerosis and paralysis agitans; and the changes described were of such a gross nature that had they been commonly present they would have been verified by more recent observations. The results of examinations by modern methods have either been negative or but slight changes have been discovered. Among the changes that have been described are hyperplasia of the connective tissue, with thickening of the walls of the finer vessels, increase of nuclei, and small islets of connective tissue which spread inwards from the surface of the cord, sclerosis of the lateral and posterior columns of the cord, pigmentation or atrophy of the anterior horn cells, and varicosity of the axis cylinders. The central canal of the spinal cord has been obliterated in some cases, and in others dilatation of the capillaries and small extravasations of blood have been found. Changes in the peripheral nerves have also been described.

In some cases no changes have been found in the central or peripheral parts of the nervous system, but the muscles have shown both hypertrophy and atrophy of fibres, with proliferation of the connective tissue.

The following are the changes that the most recent histological methods have revealed: The cells of the anterior horns, markedly pigmented, rounded, and granular; the nerve fibres in the posterior columns and peripheral nerves degenerated to some extent, the muscle fibres in part atrophied, and in part fatty or hyaline; the interstitial tissue in the cord, peripheral nerves, and muscles increased; the vessel walls thickened and the glia tissue increased. In two cases we have had the opportunity of examining the changes that were found were marked pigmentation of the cells of the brain and spinal cord, and in one of them fatty degeneration of certain muscle fibres.

The most constant changes may be said to be increase of the connective



tissue of the cord, thickening of the vessel walls, and pigmentation of the cells. Too much importance must not, however, be attached to these changes, firstly, because many cases have been examined with an absolutely negative result; and, secondly, because all the changes described have been found in the cords of old people who have not manifested any sign of paralysis agitans during life. Pigmentation of the cells is the most common change in old people, and the older the patient the more marked the pigment. Obliteration of the central canal of the spinal cord is a common feature in the cords of adults, and it is almost unnecessary to say that the same is true with regard to the thickening of the vessels. It must be admitted that so far as the morbid anatomy is concerned there is little to be said against the view held by some that paralysis agitans is merely premature senility of the nervous system.

*Pathology.*—Certain clinical facts seem to point strongly to the cerebral origin of the disease. The hemiplegic character during the earlier stages is not infrequently a marked feature, and the disease may for a considerable period remain limited to one side; and further, the progress of the disease tends to follow a course very suggestive of a cerebral origin. The character of the tremor is that associated with cerebral rather than with spinal disease, and the similarity of the tremor to that produced by fright has been called attention to by more than one author. The view taken by Dr. Hughlings-Jackson that the morbid condition lies in the cerebellum is one of great interest. He suggests that there is a loss of the normal balance between the antagonising influences of the cerebrum and cerebellum.

*Diagnosis.*—In a typical case the bent position of the body, the fixed expression of the face, the muscular rigidity, and the tremor make the diagnosis a matter of great simplicity. Even in cases where the tremor is absent the position of the patient and the rigidity of the muscles were so characteristic that a mistake can hardly occur. The tremor of senility resembles that of paralysis agitans very closely, but the characteristic position of the limbs and trunk are wanting, the head is chiefly affected, and the tremor is only brought out or is increased on voluntary movement.

From disseminated sclerosis paralysis agitans is distinguished by the later age of incidence, the attitude, the character of the tremor which is not increased on volitional movement, and by the absence of nystagmus, diplopia, optic atrophy, and affection of the sphincters.

The tremors due to alcohol, mercury, brass, and other toxic agents are distinguished by the absence of the characteristic attitude, propulsion, and subjective sensory sensations, while there is direct evidence that one or other of these toxic agents have been in operation.

From hysterical tremor it is only during the early period of the disease that any difficulty can really arise when the tremor is limited to one arm or hand, and the characteristic attitude has not developed. The tremor is more influenced by psychical phenomena, the oscillations are greater, and other signs of hysteria can be detected.

In Huntingdon's chorea the amplitude of the movement is greater and more irregular; the muscles of the face share in the movement. The characteristic attitude is wanting, and the hereditary and family history and the signs of mental impairment are sufficient to make the diagnosis clear.

In the tremor of hemiplegic origin the unilateral character, the greater rigidity, the increased reflexes on the affected side, and the history of sudden onset, would probably be sufficient to allow of a correct diagnosis.

From chronic chorea and post-hemiplegic chorea there should be no difficulty in distinguishing the movements.

*Prognosis.*—The prognosis is unfavourable; improvement and cure can only take place most rarely. The disease itself rarely causes death, but some intercurrent affection, most commonly of the respiratory system, is usually responsible for the fatal issue.

*Treatment.*—In general, rest both bodily and mental is of the first importance. In an early stage this may be attempted by rest in bed, but the patient often becomes so restless under this treatment that it should not be persisted in unless marked improvement has taken place. Patients often feel better while they are taking some exercise. Passive movements may be tried with some benefit.

Warm baths for a prolonged period have been used with good results, and the same may be said of applications of the constant current.

All manner of drugs have been tried with most varying results; the following are those most in use: belladonna, ergot, arsenic, cannabis indica, eserine, silver nitrate, strychnine, iodide of potassium, opium, hyoscine, biborate of soda, bromide salts, sulphate of duboisin, tincture of veratrum viride, and tincture of gelsemium.

Sodium salicylate may be useful in those cases in which there is much joint pain.

Of historical interest are the shaking sofa and the vibratory jacket, which were invented in response to the observation that patients were better after a railway journey and after being shaken up in a bus; they have not, however, been shown to be attended with any beneficial results.

Patients frequently adopt various methods and attitudes in order to prevent the tremor, one of the most common being to fix the hands by placing them on the knees.

In late cases special precautions have to be taken against the formation of bedsores, and the position of the patient must be frequently changed.

**HEREDITARY ATAXY (Friedreich's Disease).**—*Definition.*—An hereditary and family form of disease occurring in the first and second decades of life, the leading features of which are ataxia, nystagmus, deformities of the spine and feet, and absent knee-jerks.

*Etiology.*—The most important factor in the etiology of this disease is its tendency to affect several members of a family; not only does the malady affect brothers and sisters, but it also passes from parent to child for it may be three to four generations; certain members of a family may escape, but the disease may reappear in their children.

Other members of the same family not infrequently suffer from some nervous affection, such as insanity, epilepsy, or chorea.

There is no evidence to show that syphilis or alcohol in the parents bear any causal relation to the disease.

The occurrence of the acute specific fevers has, we believe, more influence on the onset of the disease than is generally supposed, and this factor is usually more marked in isolated cases than when many members of a family are affected.

Both sexes are about equally liable to be attacked.

The age at which the onset of the disease is most common is stated by Gowers to be between the 7th and 8th year, many cases manifest their first symptoms between 12 and 16, and a few between the latter age and 25. Some cases have shown symptoms as early as two years of age.

*Onset.*—The onset of the disease is most gradual, the first symptom to



appear being usually ataxia, then the speech becomes affected, while later the inco-ordination of the arms develops. The curvature of the spine and deformity of the feet are generally later developments, although in some cases they have been the first symptoms noticed.

The course of the disease is most uncertain; at times it seems to advance rapidly, while at others it becomes stationary, and may remain so for years, and that some remission of symptoms occurs is certain from the way in which anæsthesia will often pass off, but such remissions are never so marked as those which are often seen in disseminated sclerosis.

*Symptoms.*—The most prominent symptom in the disease is the inco-ordination that the patient exhibits when attempting to walk. In slight cases he stands with the legs wide apart, reels slightly, raising the foot rather higher than necessary, and bringing it down with a slap on the floor, while in extreme cases it may be so marked that he crosses the legs from side to side in order to save himself from falling. Even with patients who are no longer able to walk the ataxia is well shown by making them attempt to place the heel of one foot on the knee of the opposite leg.

A patient who is ataxic in his gait will often stand steadily with the eyes shut, but in other cases the unsteadiness is markedly increased when the eyes are closed.

The ataxia of the arms is usually less marked than that in the legs, and it is often only obvious when the patients attempt to feed themselves.

In children the frequent stumbling in walking and clumsy manner in which they grasp an object, and in which they feed themselves, is often attributed to carelessness or confused with chorea.

Apart from the inco-ordination exhibited on the performance of voluntary acts, there are almost constant "choreiform" movements or static ataxia as designated by Friedreich while the patient is sitting or standing. If such a patient be observed while standing, various groups of muscles in the thighs and legs are seen to be in constant movement in order to maintain the equilibrium. In the sitting posture the muscles of the trunk and shoulders are in similar action, and the head, which is often held forward, has an almost constant nodding movement, while the arms are similarly affected. So marked a feature is this movement in some cases that the affection is mistaken for chorea of a chronic type. It is worthy of note that the ataxia is frequently worse after rest and better after a certain amount of exercise.

*Ocular Symptoms.*—Nystagmus is almost a constant symptom, it may be present while the eyes are at rest, but is accentuated when they are turned to the right or left; paralysis of the ocular muscles is rare, but does occur. The pupils are generally equal, and react well to light and on accommodation; occasionally, however, they fail to respond to light, and, as Sir William Gowers points out, when this sign is present juvenile tabes should be suspected, especially if the pupil defect is associated with optic atrophy, for optic atrophy is said not to occur in Friedreich's disease. Vision is said to be occasionally defective.

Deformities of the spine and extremities are always most characteristic. The most common position assumed by the foot is that of pes cavus, the dorsum of the foot is arched, and the plantar surface is often so contracted as to form a fold across the sole of the foot, the big toe is drawn back, being flexed at the junction of the phalanges and extended at the metatarso-phalangeal joint. Although this is the common position, talipes equinus



and equino-varus also occur in about one-fifth of the cases according to Griffiths.

The prominence of the extensor proprius hallucis is often an early and marked sign, and later the tibialis posticus and the peroneus longus tendons stand out behind the internal and external malleoli respectively. The smaller toes also are in a position of extension. The foot as a whole is dropped. The deformity of the foot is explained by Duchenne as due to the weakness or paralysis of the interossei combined with weakness of the dorsi-flexors of the ankle; another explanation, however, is that it is due to an increased muscular tone in the peronei, tibialis posticus, and extensor muscles of the toes secondary to the involvement of the pyramidal system, and this is probably correct, for it has been shown by Collier that after affection of the pyramid in cases of hemiplegia or paraplegia the foot tends to assume a position of pes cavus.

Deformities of the hands have also been described, but are comparatively rare, that which is most frequently seen is the *main en griffe*, and when this occurs it is probably due to the weakness of the interossei and contraction of the unopposed extensors.

Curvature of the spine is very commonly present and may be extremely marked. It is usually situated in the dorsal region, with the concavity to the left, the central point of the curve being situated between the sixth and ninth dorsal spines; there is also not infrequently some kyphosis at the level of the upper dorsal vertebræ.

In most cases no alteration in sensation can be detected either to touch, pain, heat, or cold, and the sense of position is normal, and this may even be so when Romberg's symptom is well marked. In exceptional cases there may be anæsthesia of one or both extremities, which does not always persist; in other cases there has been loss of sensibility to heat and cold; while in others, again, there has been delayed sensation and diminution of the acuteness of sensation.

Subjective sensations are occasionally complained of, numbness of the hands and of the soles of the feet, and these are often associated with coldness and blueness of the extremities. Lightning pains have been noted in rare instances, but visceral crises do not occur.

The muscles react to faradism, although they not infrequently require a stronger current than normal, and it has also been noted that the patient will bear without any discomfort a far more powerful current than can be borne by a healthy individual. No polar change is demonstrable with the galvanic current, although there may be some diminution in the response. Myotatic irritability of the muscles is lost.

Some defect of articulation is nearly always present, the words are jerked out and interrupted by sudden and irregular pauses, and they are slurred over so that the ends of the longer words become clipped, and the first letter often elided, the general effect being that the speech is slow and drawling.

The movements of the palate and larynx are normal, although abductor palsy of the cords has been noted. Mastication and deglutition are normal; there is at times some difficulty in retaining the saliva.

It is generally stated that in the majority of cases there is no mental defect, and this is no doubt true; on the other hand, it is not uncommon to meet with marked mental defect. The expression of the patient, together with the slow utterance, often suggests some mental impairment which is not borne out on examination; but, quite apart from any such error, not a few cases exhibit marked mental defect. It is difficult to



estimate the exact proportion of cases in which such defect occurs, but Griffiths gives the number as 21 out of 143 cases; he, however, qualifies the statement by saying that in most of these the affection was more than questionable.

The superficial reflexes are usually present and brisk, the knee-jerks are absent, although in exceptional cases they may be present, and even exaggerated, and in these latter cases ankle clonus may also be obtained, as in a case recently reported by Gladstone in *Brain*, 1899.

The plantar reflexes, when they can be obtained, give a definite extensor response. The arm-jerks cannot as a rule be obtained.

There is no incontinence of urine or fæces, the act of micturition, however, may be somewhat delayed. Sexual power may be lost, but as the disease frequently commences before puberty, it is difficult to state how often this occurs. Menstruation is also often delayed.

*Pathological Anatomy.*—The brain and cerebellum usually appear normal to the naked eye, the spinal cord is smaller and shrunken, and lies in a loose dural sac which contains an excess of cerebro-spinal fluid. The pia-arachnoid over the posterior columns is thickened and adherent to the posterior column of the cord. On section of the cord the gray and translucent appearance of the sclerosed posterior columns is obvious to the naked eye, and the posterior roots appear very small, the anterior roots are normal.

Examined microscopically, there is degeneration both of the lateral and posterior columns, that in the posterior columns extending from the sacral region to the posterior column nuclei in the medulla, the column of Goll being more affected than that of Burdach. In the lumbo-sacral region of the cord the endogenous fibres in the posterior columns, that is to say, the septo-marginal tract and the cornu-commisural zone are left unaffected. Lissauer's tract is commonly affected, and Clark's column is also involved. In so far, then, as the posterior columns are concerned, the lesion corresponds very closely to that found in tabes. Besides the affection of the posterior columns there is degeneration in the direct cerebellar tract, and also in the ascending antero-lateral tract of Gowers. The crossed pyramidal tracts show marked degeneration in most cases, but the degeneration in others is slight, and has been regarded not as that of the efferent fibres, but as that of afferent fibres. In advanced cases, however, the pyramidal tracts are very markedly affected, and in rare cases the direct pyramidal tract may also be involved.

Although the degeneration is most marked in the tracts above named, yet it is not limited to them, but tends to involve a good deal of the periphery of the cord. There is always a considerable amount of connective tissue replacing the fibres that have undergone degeneration. There is frequently extensive degeneration of the fibres entering the posterior horn.

In a few cases changes have been found in the cells of the anterior horns. The posterior roots between the ganglion and the cord show extensive degeneration. In these roots some very fine nerve fibres are often present, but by far the greater part of the root is composed of empty sheaths and connective tissue; these fine fibres have been considered to be embryological in type. The roots in the lumbar region generally show more change than those either in the dorsal or cervical region.

The posterior root ganglia appear smaller than normal, the cells are small and pigmented, and the finer granules of the cells stain badly.

There is marked degeneration of the fibres that enter the ganglion from



the posterior root, while those of the anterior root in close proximity to the ganglion are normal, with the exception possibly of a few degenerate fibres.

In the mixed nerves the motor fibres are normal, while those coming from the posterior root ganglia are atrophied.

The peripheral nerves show considerable degeneration, and it would seem that the portions more peripherally situated suffer more than those nearer the cord.

In the medulla the degeneration can be traced up to the funiculus gracilis and cuneatus, the direct cerebellar tract is markedly affected, and the ventral cerebellar tract to a lesser degree.

Some degeneration may be seen in the pyramids as high as the medulla.

The nucleus gracilis is practically absent, and the nucleus cuneatus very poorly developed.

The cerebrum and cerebellum usually appear normal on microscopic examination. It is not uncommon to find some developmental abnormality in the cord.

*Pathology.*—That the affection of the posterior columns closely resembles that found in tabes there can be little doubt, but various views have been held as to whether the condition is due to arrest of development or to an early degeneration. All the evidence from the clinical side of the question, however, points to the latter view as that which is most probably correct. It is difficult to believe that an undeveloped cord could perform its functions for many years before showing signs of failure. The curious manner in which several members of a family are affected, however, points to some inherent tendency to failure as soon as any extra strain is thrown upon the nervous system.

Although many of the symptoms are accounted for by the pathological condition found, yet there are others which cannot be so explained, such as the nystagmus, the defective articulation, and the choreiform movements.

Some authors have looked upon the disease as essentially cerebellar, but the pathological evidence is almost entirely against such a view, in that the cerebellum in almost all undoubted cases of the disease has been found to be normal. There seems to be no doubt that the affection frequently follows some acute disease, and this is possibly more often the case in the isolated cases than in those which occur in families; the acute disease can, however, only be regarded as an exciting cause acting on a nervous system which for some reason is congenitally weak.

*Diagnosis.*—In a marked case in which there is ataxia, nystagmus spinal curvature, and deformity of the foot, and in which the knee-jerks are absent, there should be no difficulty in diagnosing the disease. In other cases the affection may be confused with disseminated sclerosis, in which disease, however, the intention tremor is much coarser and more jerky, the nystagmus is more marked, the knee-jerks are exaggerated, ankle clonus is present, and ocular palsies, diplopia, and optic atrophy are not uncommon. The age of the patients is different, for disseminated sclerosis commonly begins during the third decade of life. In certain cases in which anomalous symptoms are present the diagnosis is by no means so easy, for it has already been pointed out that exaggerated knee-jerks and ankle clonus may occasionally be present in Friedreich's disease.

The diagnosis from a typical case of tabes presents no difficulty, but from the juvenile form of tabes and from the associated disease of tabes and general paralysis occurring in the subjects of congenital syphilis, considerable difficulty is likely to arise. The presence of optic atrophy,



irregular-sized pupils, the Argyll-Robertson phenomenon, and bladder trouble, together with the absence of spinal curvature and foot deformity, and nystagmus, would all be in favour of the diagnosis of juvenile tabes. From a cerebellar tumour the slow and gradual onset, the absence of headache, vomiting, and optic neuritis, together with the curious variability in the knee-jerks commonly seen in cases of cerebellar tumour, should render the differential diagnosis simple.

Hereditary cerebellar ataxia (Marie's disease) is later in its onset (after the age of 20), the mental affection is more marked, there is optic atrophy, the knee-jerks are active, and the ataxia is greater.

To chorea of a chronic type the disease only bears a superficial resemblance, and apart from the character of the movements and the ataxia, the curvature of the spine, the deformity of the foot, and the presence of nystagmus, are usually sufficient to form a diagnosis.

*Prognosis.*—The prognosis is unfavourable, although the disease does not necessarily advance, yet it leaves the patient in an enfeebled condition, so that he readily falls a victim to some intercurrent affection.

*Treatment.*—Drugs have no effect either to arrest or to cure the disease. Massage and electricity may be of some service. The patient should be encouraged to take moderate exercise, and should perform various co-ordinate movements both with the arms and legs.

### FUNCTIONAL PARALYSIS

Inasmuch as our knowledge of the intimate nature of functional disease is incomplete, the definition of "functional paralysis" is difficult and unsatisfactory, and the conditions which are to be included under this term are separated from conditions not so included by a somewhat arbitrary line. "Functional paralysis" is a term applied to a natural group of cases characterised by the rapid onset of paralysis, often from trifling causes. The paralysis after a longer or shorter time often disappears rapidly, sometimes instantaneously. The nature of the paralysis is frequently such as to render its explanation by any conceivable organic lesion difficult or impossible. While the signs and symptoms commonly met with in organic disease are absent, certain peculiar symptoms, characteristic of hysteria, are often present. No organic disease which could account for such symptoms has been found.

There is no single symptom or physical sign the presence of which proves the nature of any given case of paralysis to be functional. On the other hand, there are many physical signs and symptoms the presence of which absolutely exclude from the category of functional paralysis. The failure of pathological investigation to reveal any morbid changes in the organs does not justify the inclusion of a disease into this group, for the term is here used arbitrarily for a clinical group, and not in its true etymological sense. As examples, epilepsy, chorea, and paralysis agitans, which are distinct from the class of diseases here described, are without discovered morbid lesions, and the two first are eminently "functional" diseases in the etymological sense of the word.

There are, further, two important considerations in the recognition of functional paralysis. First, that long-standing functional paralysis sometimes leads to degenerative changes in the elements of the nervous system supplying the affected region, and such degenerative changes may be progressive. When these take place the clinical picture may acquire an organic aspect, and the autopsy reveal gross tissue changes. Secondly,



functional paralysis may be not infrequently an early symptom of organic disease, especially of the disease disseminated sclerosis; it is then, as a rule, accompanied by some distinctive organic sign.

*Etiology.*—The disease is much more common in females than in males, and most frequently occurs in young adult life, from the time of puberty to the age of 35 years. It has occurred as early as the fifth year, and in females is sometimes first manifested as late as the time of the menopause. Neuropathic heredity is frequent. Among parents and relations of the patient are often found hysteria, epilepsy, insanity, chorea, and “nervousness.” The disease is more common in Latin than in Saxon races, and a fair proportion of cases occur amongst the Jews. The patients themselves frequently present signs of a neuropathic tendency. In children precocity and mental instability or impairment are often present. In adults, often the symptoms of hysteria are present, and there is always some mental instability. Some of the cases, after years, become mentally deranged, and occasionally organic nervous disease in some form appears, presumably from the same neuropathic tendency to which the functional symptoms are attributable.

Predisposing causes are often absent, but in some patients a cause for general deterioration of health is evident, and this may be any acute disease, especially influenza, anæmia, dyspepsia, privation, long hours, and overwork. By far the most important exciting causes are mental emotion, shocks, and physical injuries, the latter being often slight. Long-continued anxiety, disappointments, money losses, bereavements, sudden frights, railway, and carriage accidents are frequently the immediate precursors of the paralysis. In the recent South African war several cases arose from slight bullet wounds. The disease is sometimes attributable to pelvic trouble, especially where this has required much local treatment. In cases where symptoms first arrive in middle life, alcoholism, and in women the menopause, are probable important factors.

*Clinical Aspect.*—Functional paralysis may affect almost any part of the voluntary muscles of the body. The result may be slight or severe paralysis, but rarely complete paralysis as regards a whole limb. In the slight forms there may be weakness with ataxy or with tremor. In the more severe forms the paralysis may be flaccid or spastic, and in the latter case there are usually contractures. It is impossible to describe functional spasm separately from functional paralysis, as where spasm exists there is paralysis in effect.

The distribution of functional paralysis is either hemiplegic, paraplegic, or monoplegic. In this country functional paraplegia is the more common.

**FUNCTIONAL HEMIPLEGIA.**—The paralysis affects one side of the body, more frequently the left, and may be flaccid or spastic. The arm and leg may be uniformly affected, or the affection of the leg may preponderate, but the face is never affected in flaccid palsy and seldom in spastic palsy. In the latter case there may be contracture of the face and contracture of the tongue on protrusion, so that that organ is held with its non-affected side transversely between the lips. The neck and trunk muscles are rarely affected. In the slight flaccid cases there may be simply general weakness with capacity to perform each movement; in the more severe cases there may be inability to raise the limb off the bed. The paralysis may be complete, but for the movement of one muscle, and the movement of that muscle may be normal. For instance, a woman with complete flaccid paralysis of the left leg, without wasting, and with a normal knee-jerk and electric excitability, was always able to extend the great toe. She could perform this act with great rapidity. Such an act was incompatible with any organic lesion



causing otherwise complete hemiplegia, and the patient after many months recovered suddenly. The importance of such contradictory symptoms in functional paralysis is great, as they occur frequently. In the spastic cases there may be contracture of the face, tongue, and muscles of mastication on the affected side. Sometimes there is a degree of torticollis. The spasm is usually equally resistive to passive movement in all positions of the limb—there is no “clasp-knife” phenomenon, and the fingers when affected do not relax on passive flexion of the wrist as in organic hemiplegia. Passive movement is as a rule much objected to by the patient. The spasm when severe and without intermission is termed contracture, and when this occurs the limbs assume characteristic positions. In the upper limb the arm is adducted, the elbow flexed, and the fingers either flexed at all joints or extended at the inter-phalangeal joints and strongly adducted (interosseal position). The lower extremity is always extended at the hip and knee, often adducted and rotated in at the hip; the foot is dropped and inverted. Contracture of any degree of severity does not relax during sleep, and only relaxes in the late stages of surgical anaesthesia.

A peculiar characteristic of both types is that when an attempt is made to voluntarily or passively move a joint in a particular direction, the antagonistic muscles at once contract. Where voluntary movement is possible it is generally tremulous, and this is especially evident when power is returning and spasm lessening. The tremor may be the most conspicuous symptom of the hemiplegia. Where the patient is able to stand, the gait is characteristic, the paralysed leg being dragged helplessly behind the other, the dorsal surface of the toes resting upon the ground. Where there is some degree of power in the lower extremities, the gait is as if the patient voluntarily exaggerated all the difficulties, and the most grotesque movements result, which have no place in the clinical picture of organic hemiplegia. There is no muscular wasting except in cases of long duration, and then it is only slight. The electrical excitability of the muscles is unaltered. Slight vaso-motor disturbance may be present in the regions where contracture is present, and is probably due to pressure upon the veins by the spastic muscles. Slight trophic changes in the periphery of the limbs are met with, and rest adhesions may form in the joints. The deep reflexes are exaggerated; more upon the affected side where foot clonus may be present. The plantar reflex is often absent on one or both sides. Where present it is always of the flexor type. The abdominal reflex is often exaggerated upon the paralysed side.

The other signs of functional disease, often present in functional hemiplegia, are hemianæsthesia to all forms of sensibility, and especially to pain, involving the body and frequently the mucous membranes up to the middle line. The muscular sense is sometimes retained. Unilateral loss of smell, diminution of hearing, and crossed amblyopia and hysterical convulsions are often present (*vide* “Hysteria”).

The three following physical signs are of great value in diagnosis:—(1) The plantar reflex, which is always flexor when present in functional disease, is almost always extensor in organic disease, where the pyramidal tracts are involved. (2) On pressing the chin downwards against a firm resistance the head deviates to the sound side in organic hemiplegia; it does not deviate in functional hemiplegia. (3) The patient is placed supine upon the bed and the legs uncovered. She is then directed to sit up suddenly. When this act is performed by a normal person, both thighs and knees are flexed slightly; during the act in organic hemiplegia, where any rigidity is present, the paralysed thigh moves up much more than the sound. In



functional hemiplegia, where rigidity is present, the affected limb does not move at all or only slightly. (Babinski's sign.)

It is here convenient to place in the form of a table the chief features serving to distinguish functional hemiplegia from organic hemiplegia.

	Functional.	Organic.
Vision . . . . .	Crossed amblyopia	Hemianopia
Hemi-analgesia . . . . .	Frequently complete	Never complete
Face . . . . .	Never paralysed	Often paralysed
Paralysis . . . . .	Rarely complete	Often complete
Gait . . . . .	The foot is dragged behind	Leg moved forward in a half circle
Contracture of knee and foot	Knee extended. Foot, equino-varus	Knee, often flexed. Foot, pes cavus
True foot clonus . . . . .	Seldom present	Generally present
Abdominal reflex . . . . .	Often exaggerated	Diminished or absent
Plantar reflex . . . . .	Absent or flexor	Extensor
Inclination of head to sound side on depressing chin	Absent	Often present
Babinski's hip phenomenon where rigidity is present	Absent	Present

FUNCTIONAL PARAPLEGIA.—The onset of the paralysis is almost invariably sudden, and often follows some fright or emotion. The loss of power is usually moderate in degree at first, and gradually increases, and may assume any degree from slight ataxy of movement to complete paralysis. The power which remains is put into action irregularly. There is often resistance to passive movement. If an attempt be made to execute a given movement, opponents of the acting muscles may be felt to contract, and any resulting movement is accompanied by tremor. Often the nature and distribution of the paralysis are peculiar; individual movements and muscles may be exempt in cases where the paralysis is otherwise complete. A patient who, while in bed, may perform all movements against moderate resistance may be entirely unable to make even an attempt to stand or walk; in other cases standing may be possible, but on the attempt to walk the knees immediately give way and the patient falls to the ground. This is the condition to which the term *astasia abasia* has been applied (*vide infra*). It is not peculiar to functional disease, but may exist in any case where there is loss of power or of the muscular sense in the quadriceps extensor femoris, peripheral neuritis, tabes dorsalis, spastic paraplegia, disseminate sclerosis, etc. It is explained by the fact, that with the knee extended very little muscular effort is required to keep the knee rigid for the support of the body weight, but directly the knee is bent the whole weight of the body is thrown upon the quadriceps.

Further, where paralysis approaches completeness in the waking state, a patient may, if carefully watched, be seen to move the affected limbs freely when in the lighter stages of sleep, and the same result is often seen in the early stages of surgical anæsthesia.

Such contradictory phenomena occur in great variety in functional paralysis, and though highly characteristic of the disease, are not absolutely pathognomonic, as they also occur, though rarely, in organic paralysis.

When walking is possible the gait is often characteristic, and has been termed by Hughlings Jackson the "constructed" gait, for every difficulty is exaggerated, and all kinds of peculiar movements of trunk and limbs added, while the patient often evinces much emotional disturbance during the attempt to walk. Sometimes a patient who walks in this manner when unsupported may walk quite naturally if she is allowed to hold a single



finger of the observer, this slight support giving her confidence. It has been stated that such patients never fall, when walking, in such a way as to hurt themselves, but collapse gently on to the ground, and this fact has been used as a test to distinguish functional paraplegia. It is, however, not invariably true, and as a test it has been fraught with severe injuries and dangerous consequences.

The paraplegia is sometimes quite flaccid, generally slightly spastic, not often severely spastic, and the occurrence of contractures is much less common than in functional hemiplegia. When contractures occur they resemble those described in functional hemiplegia. There is never flexor contracture at hip and knee such as commonly occurs in organic spastic paraplegia; and pes cavus, so characteristic of the latter condition, never occurs. Spontaneous clonus (epileptoid spasm) never occurs. In most cases the paralysis is confined to the lower extremities, and these may be unequally affected. Rarely all four extremities are affected. The trunk is sometimes affected, but never segmentally; there is then inability to rise from the supine position. There is no muscular wasting, and the electric excitability is unaltered. The tendon reflexes are increased, and there may be foot clonus. Sometimes this latter is indistinguishable from the foot clonus of organic disease, but more frequently it is commenced by a voluntary movement of plantar extension at the ankle, and does not occur if the foot be fully dorsiflexed (such a clonus may be produced at will by most healthy people in certain positions of the leg and foot, for instance sitting forward with the feet on tiptoe). The plantar reflexes are often absent, whether anæsthesia be present or not. When obtained they are always of the flexor type. Sometimes a movement of the tensor vaginae femoris alone occurs on stimulating the sole of the foot.

Incontinence of the sphincters never occurs. There may be slight difficulty in starting the act of micturition, an exaggeration of a condition not uncommon in healthy persons. Since incontinence of sphincters is one of the most important symptoms in distinguishing functional from organic disease, patients must be very accurately and directly questioned, more especially as in women slight difficulties are often concealed. As examples of the care necessary to avoid erroneous diagnosis, the following instances may be cited:—A patient with a well-marked functional paraplegia complained of inability to hold her water. She was admitted into hospital and was found to be suffering with thrombosed hæmorrhoids, the treatment of which cured the incontinence. Another patient, in whom precipitancy of micturition had led to the diagnosis of organic disease, had suffered with sphincter trouble ever since infancy, and the paraplegia had occurred from shock six weeks before examination. In a third case severe cystitis and consequent incontinence had been set up from the repeated use of an unclean catheter by the patient herself, the catheter having been originally ordered for hysterical retention.

It is the rule for anæsthesia to be present in some form in the paralysed region. Sensibility to pain is as a rule most impaired, and that to touch least, but the anæsthesia may be complete to all forms. The muscular sense is sometimes absent, sometimes preserved. The anæsthesia is distributed as a rule in "stocking or glove fashion," frequently it is limited above by Poupart's ligaments and the iliac crests, and the perineum is almost always exempt, even when the anæsthesia extends on to the trunk. Hemi-anæsthesia may be present in addition to paranæsthesia. Slight trophic changes in the skin of the periphery, rest adhesions in the joints, and slight vaso-motor palsy may be present.



*Functional Monoplegia.*—The general description above given for hemiplegia and paraplegia apply to functional paralysis when one limb only is affected. Special importance must be attached to the fact that the anæsthesia is apt to be much more extensive than is the paralysis. Thus in a case of functional paralysis, limited to the forearm and hand, complete analgesia involved the whole upper limb.

*Functional Ptosis.*—This condition is usually bilateral, and differs from true ptosis in that the eyes are completely closed, and that an attempt to raise the lid with the finger is resisted by contraction of the orbicularis. There is no paralysis of the eye muscles, and the reflex action of the pupil is normal. There may be occasionally dissociated movements of the eyes.

*Functional Spasm of the Palate.*—This is usually an intermittent spasm, always involving the side of the pharynx, and sometimes also the vocal cord. Its occurrence is rare.

*Functional aphonia* is one of the commonest forms of functional paralysis. Laryngoscopic examination reveals complete or partial adductor paralysis. Abductor paralysis never occurs as a functional condition. Occasionally all the movements of articulation and phonation are weakly performed.

*Hysterical Mutism.*—See “Aphasia.”

Phantom tumour, which may occur in connection with functional paralysis, is apparently due to contracture of the lateral muscles of the abdominal wall with paresis and relaxation of part of the rectus abdominis muscle.

*Astasia Abasia.*—This term denotes a symptom and not a disease. The phenomenon is met with in hysteria, and less frequently in other nervous affections, and is most often evoked by emotional disturbance or treatment, though it has also followed infectious diseases.

The patient while able to perform all movements of the legs freely and with good power while lying in bed, becomes helpless, and collapses on trying to stand or walk, or experiences great difficulty in walking.

See also “Hysteria” and “Neurasthenia.”

*Course and Prognosis.*—The course of the disease is very varied, and is often marked by partial recovery, followed by relapses. The paralysis may disappear suddenly, sometimes as the result of some impressive therapeutic measure, sometimes without any definite cause. This result is more common in cases where the duration of the paralysis has been short. Much more often gradual improvement takes place, and the paralysis takes weeks or even months to disappear. The prognosis is worse, as regards ultimate recovery and the occurrence of relapses, when the general signs of hysteria are well marked. It is also worse the longer the duration of the disease, for the long-continued functional derangement must cause molecular alteration of the nervous elements. Complete and lasting recovery, however, has occurred after almost complete paraplegia has been present for seventeen years. Most of the cases ultimately recover so far as the paralysis is concerned. Some few end in organic disease of the nervous system, and death has occurred in rare instances from the anorexia and vomiting. The prognosis is favourable in proportion as definite causes can be traced and removed, especially deterioration of health of remediable character.

*Treatment.*—The treatment of functional paralysis is that of hysteria in general (*q.v.*); the Weir Mitchell treatment being especially valuable. As regards the special treatment of functional paralysis, it is important that the patient should expect gain in power to follow any treatment adopted. Faradism to the affected limbs, passive movements, and massage are the



necessary remedies. Electricity, which these patients as a rule find very disagreeable, should be applied by the medical attendant or by a skilled nurse, as the more profound is the patient's respect for the treatment the better is the result. In severe cases, when movement is returning, the calls to increased effort must be very gradual and systematic, care being taken that no attempt is allowed to fail entirely or to produce fatigue. A patient who has been long bed-ridden should be encouraged to crawl about at first. When walking is first attempted it should be with the aid of a person on either side, subsequently a steady frame on wheels, which the patient pushes along and uses as a support, should be used. Any measure producing severe pain is harmful. Static electricity and circular blisters round the limb are useful. Sometimes brief ether anæsthesia has removed contracture. In long-standing cases, where contracture has led to permanent shortening of the muscles, tenotomy is sometimes advisable, but only after the disappearance of the paralysis.

*Diagnosis.*—In the differentiation of functional paralysis from that of organic origin, it must be borne in mind that organic disease simulates functional disease more often and more closely than does functional disease organic disease. A case in which, after careful consideration, much doubt arises, is probably organic. Always to be kept in view is the possibility of the appearance of true organic disease upon a functional background. Speaking generally of functional paralysis, the age, sex, and temperament of the patient must be given weight. The history of previous manifestations of hysteria and the adequacy of cause to result in the production of the symptoms must be considered. In functional hemiplegia the absence of the usual causes for apoplexy (*morbis cordis*), etc., and the absence of conjugate deviation, true convulsion, coma, and aphasia at the onset are important. Crossed amblyopia, complete hemianalgesia, and the occurrence of contracture soon after the onset, are the most important positive signs that a hemiplegia is functional. Absence of facial involvement is a useful negative sign. The importance of the plantar reflex and of Babinski's sign have been already dwelt on.

The diagnosis of functional paraplegia is more difficult, since it is closely simulated by the early stages of one type of disseminated sclerosis. The most important positive functional signs are contradictory muscular phenomena, the gait, contractures and anæsthesia of greater extent than the paralysis, its upper limit being a circular line round the limb (almost the only other condition in which "stocking" and "glove" anæsthesia exist is peripheral neuritis—the anæsthesia is here always incomplete, always of smaller area than the paralysis, and is usually accompanied by pain).

The Argyll-Robertson pupil, ocular paralysis, optic atrophy, incontinence of sphincters, girdle pain, alteration of faradic excitability, loss of knee-jerk, and the extensor response in the plantar reflex, are signs, the presence of one of which alone excludes uncomplicated functional disease.

### Paralysis, Insanity associated with.

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DISEASE OF THE BRAIN . . . . .	187		

GROSS disease of the brain, and of its hard and soft coverings, is a frequent cause of mental aberration. In infancy and the early stages of life it



arrests mental development and produces various forms of idiocy and imbecility, associated or not with epilepsy. Degenerations of the brain and of its membranes also occur frequently as secondary symptoms in many forms of insanity. In the present article consideration is limited to those grosser tissue changes which are the primary cause of insanity in adult life.

First among these to be dealt with are sclerotic affections of the neuroglia and blood-vessels of the brain. It must be admitted that our knowledge of the ætiology and symptoms of cerebral sclerosis is not so accurate as could be wished. This is probably because many of the diseases referred to are comparatively rare, and because their symptoms, even when recognised, are often vague and indefinite.

Sclerosis of the brain is of two kinds, primary and secondary. Secondary sclerosis, or atrophy, is consequent upon some previous lesion, such as hæmorrhage, and need not be separately alluded to. Primary sclerosis may be divided into two classes: (*a*) disseminated or insular, and (*b*) diffuse sclerosis.

Disseminated sclerosis is comparatively rarely met with in association with mental disease. In those cases, however, in which the invasion of the brain by the sclerotic masses is extensive there is observed, in addition to the physical symptoms of progressive muscular weakness, jerky incoördination and nystagmus, the following mental symptoms: progressive mental weakness, loss of intelligence and disturbance of the thought processes, but without any other active or positive symptoms of insanity. Gradually the symptoms end in dementia, with stupidity and loss of will power. Some cases may exhibit recurrent attacks of depression or excitement, which are, however, probably coincidental, and not connected with the disease. Certain cases may present such a striking resemblance to general paralysis as to render the differential diagnosis a matter of considerable difficulty, but the character of the tremor, of the speech affection, of the mental symptoms, and the presence of nystagmus, is usually sufficient to determine the difference between the two diseases.

Our knowledge of diffuse sclerosis is scanty, and the groups of diseases known under that name are badly defined. It occurs as a primary condition in children who are the subjects of congenital syphilis. It has also, according to Gowers, been observed, though rarely, in chronic alcoholism and associated with insular or disseminated sclerosis. Kraepelin and Homen describe the symptoms of diffuse sclerosis of the brain due to hereditary syphilis. The disease begins early in youth with vertigo, headache, unsteadiness of gait, and progressive deterioration of the mental processes. Later on is added slurring and difficulty of speech, spasms, contractures, incontinence, difficulty in swallowing, slight tremor, and in some cases convulsions. The mental weakness becomes more and more marked, and death takes place after the lapse of a series of years. The post-mortem indications usually found are endarteritis. Atrophy of nerve fibres, especially in the fore-brain, slight changes in the pyramidal cells, and some increase of the neuroglia elements are also present.

Binswanger has described a form of diffuse sclerosis which he calls arterio-sclerotic brain degeneration (*Arterio-sklerotische Hirnentartung*). This disease is, however, not confined to the brain vessels, but simultaneously affects those of other organs, such as the kidney. The vessel walls are partly atrophied, partly thickened, and the lumen is widened. Sometimes aneurysmal formations and capillary ruptures are met with. The neuroglia is increased, especially in foci around the affected vessels, and



degenerative processes are found in the nerve cells and fibres of the cortex. The disease, which appears about forty or fifty years of age, is slow in development, and the clinical mental symptoms are various. They include headache, giddiness, excitement, and thought disturbance, and during the frequent remissions in these symptoms there is observable a blunting of the intelligence and stupidity which ends in dementia. The physical signs are paretic speech disturbance, aphasic forms, and frequently permanent paralysis of various parts of the body. Another form of diffuse sclerosis of the brain has been named by Binswanger chronic progressive subcortical encephalitis. This is a disease coming on about the fiftieth or sixtieth year, and is characterised by local paralyses and progressive dementia. The pathological changes observed are atrophy of the medullary subcortical fibres, widening of the brain cavities, especially the posterior ones, and atheroma of the blood-vessels. In all forms of mental disturbance casually related to cerebral sclerosis the prognosis is grave.

*Cerebral Hæmorrhage.*—In many instances where the onset of the apoplectic condition is sudden, consciousness is lost, and a state of coma prevails. It sometimes happens that the patients on awaking from the coma become delirious. The delirium is characterised by noise, shouting, and struggling. Other patients manifest mental confusion, loss of personal identity, loss of the sense of locality, and delusions. Where there is no extensive cerebral lesion or atheroma of the arteries, most of the cases recover sooner or later, generally sooner from the foregoing symptoms, but in others a certain amount of mental weakness remains permanently. As a rule this mental enfeeblement is slight and affects only memory for dates and past events, or it produces in addition some facility of will power, and perhaps an increased emotionalism. In others, however, where there is extensive subsequent or coexisting lesion, such as laceration of the brain substance, it may either quickly or gradually terminate in a state of complete dementia. In hæmorrhagic cerebral affections, the mental symptoms, like the bodily, depend largely upon the site of the lesion, its extent, upon its character, *i.e.* its complications, and upon the rapidity of absorption of the clot. If the hæmorrhage is followed by local softening, the results are more grave. The age and state of health of the patient, and above all the state of his arteries, exercise great influence upon the subsequent course of the disease, and consequently upon the character of the mental symptoms. When there is marked aphasia the gravity of the mental symptoms is apt to be exaggerated, especially in amnesic and paraphasic conditions, where the patients talk nonsense while retaining an otherwise valid intelligence. But in senile cases with degenerated arteries all forms of mental disturbance, mania, melancholia, and delusions with hallucinations may occur. Some of the cases manifest recurrent or alternating forms of insanity, and may pass through numerous phases of mental aberration. The dominating mental state of such patients is, however, dementia with a noisy emotionalism. They are often very troublesome and difficult to manage, and are, as a rule, sleepless, especially during the acuter phases of the malady. Fortunately many of them settle down into a quiet even dementia without excitement or much depression.

The diagnosis and prognosis depend entirely upon the underlying cerebral lesion, its seat, extent, and character. In otherwise healthy persons the acuter symptoms which sometimes manifest themselves in the early stages may completely pass off, leaving, or not, a slight degree of mental weakness or instability.

*Softening of the Brain.*—The differential diagnosis between cerebral



embolism and cerebral hæmorrhage is often a matter of great difficulty. Owing to the fact that the most common seat of both lesions is the middle cerebral artery and its branches, the symptoms may be at first very similar. Initial loss of consciousness in embolism is less frequent than in hæmorrhage. In embolism due to syphilitic disease, for example, where the plugging of an atheromatous artery is a slow process, consciousness is more frequently preserved than lost. Convulsions very frequently accompany the onset in embolism. Delirium may take the place of loss of consciousness, and in atheromatous softening a brief initial loss of consciousness is often succeeded by quiet delirium lasting for several days or even weeks (Gowers). Owing to the fact that the cortex supplied by the middle cerebral is frequently implicated, aphasia is much more common in softening than in hæmorrhage. Mental symptoms are undoubtedly more prevalent and more pronounced in softening than in hæmorrhage, because when due to atheroma there is usually an extensive implication of all the cerebral arteries, which prevents reparative processes and interferes with brain nutrition, and because foci of softening are generally numerous and widely spread. The mental symptoms in brain softening caused by occlusion of an artery, especially of the middle cerebral or one of its principal branches, are generally of a chronic and persistent character, their gravity depending upon the area and extent of area of the brain implicated, and upon the state of the brain and its arteries.

*Tumours.*—The mental symptoms accompanying brain tumours depend upon their site, their size, and the rapidity with which they grow. In the larger tumours the character of the symptoms is influenced more by their pressure upon the brain, and less by their site. It has been held by some authorities that tumours of the occipital lobes are more detrimental to mental activity than tumours of the frontal lobes, and the contrary opinion is held by others. On that point, however, we have as yet no clear knowledge. In slow-growing tumours of considerable size, even when the cortex is largely implicated, the mental symptoms may be very slight up to the end. On the other hand, when the tumour by rapid growth gives rise to marked pressure symptoms, there is usually observable a varying degree of mental dulness and stupidity. The attention of the patients in such cases becomes more and more difficult to rouse, until at last, except for the pain of the headache from which they suffer, they appear indifferent to all phenomena, either external or internal. When, on the other hand, the tumour is small and of slow growth, a great variety of elements would appear to complicate its relation to insanity. According to some authorities, there may occur moral changes with obsessions and impulses towards various antisocial actions, chief among which is violence. Without questioning this observation, it may be pointed out that many such tumours are of syphilitic origin, that many of the subjects are prone to indulgence in alcohol, and that some of them suffer from epilepsy. Apart, however, from these exceptions, it may probably be admitted that not a few cases owe a change of disposition in the direction of moral decadence and impulsive conduct directly to the presence of cerebral tumour. A group of cases exists in which the presence of tumour in the brain gives rise to acute insanity, with hallucinations and delusions which bear a striking resemblance to similar symptoms of toxic origin. The most commonly met with symptoms in cerebral tumour are confusion of thought and dulness of expression. This slowness in expressing ideas is materially increased by the peculiar speech of the majority of the patients. The words are slowly and deliberately uttered, and the syllables scanned. In addition to the above symptoms there are commonly met with loss of memory, depression alternating with



excitement, and emotional instability. A noisy delirium with hallucinations and delusions may occasionally be observed. Finally, complete dementia, generally accompanied with periodic convulsions, is met with in tumours of the frontal lobes. It is well to bear in mind that hysteria may be a prominent symptom of cerebral tumour in predisposed persons, and may so obscure the physical and mental symptoms as to cause the diagnosis to be at fault (Gowers).

*Cerebral abscess*, apart from its site or size, may cause acute insanity. It is true that beyond a certain amount of confusion, due to constitutional influences, some patients offer no mental symptoms indicative of cerebral change. In acute abscesses, especially those of traumatic origin, the mental dulness and confusion are prominent symptoms. The patients are oblivious of their surroundings, answer questions at random or incoherently, they are listless, restless, dreamy, and often agitated. In addition to physical symptoms depending on constitutional disturbance and local pressure the patients may manifest catalepsy, aphasia of different kinds, cortical epilepsy, Cheyne-Stokes breathing, and other irritative symptoms. In chronic abscess the mental symptoms of the latent period are usually obscure and unimportant, and when more pronounced are generally of the melancholic type. The latent stage in such cases is, however, generally terminated by such symptoms as stupor, delirium followed by coma, or by severe mental depression ending in coma.

LITERATURE.—Kraepelin *Psychiatrie*: CLOUSTON. *Mental Diseases*.—GOWERS. *Diseases of Nervous System*.—ROBERTSON. Tuke's *Dict. of Psychol. Med.* Art. "Post-Apoplectic Insanity."—ROSS. *Dis. of Nerv. System*.—GILLES DE LA TOURETTE. *Malad. de system nerv.*

### **Paramyoclonus Multiplex.** See SPASM.

### **Paranoia.**

SYN.—*Delusional Insanity, Monomania, Megalomania, Délire de persécution, Délire des dégénérés, Verrücktheit, Chronischer Wahnsinn.*

THE term "paranoia," introduced by the German physician von Gudden, is not a convenient one either from the etymological or descriptive standpoint, but its very general acceptance in Germany and in America renders its change undesirable in the interest of uniformity. The nomenclature of the various groups of mental affections which it embraces is so diverse, and the ambiguity arising from the apparently contradictory views of numerous writers is so confusing, that a brief résumé of the historical development of our knowledge of the subject is necessary for the purpose of correctly understanding it.

The disease was first called monomania by Esquirol, who defined it as "a chronic cerebral affection without fever, and characterised by a partial lesion of the intelligence, the affections, or the will." Prichard cut through Esquirol's definition by describing two distinct affections—moral insanity and monomania. The effect of Prichard's writings on European opinion was profound, and his views are still regarded as final by many of his countrymen, for he was the first and the last English psychiatrist to contribute essentially to the knowledge of the subject. In 1852 Lasègue published his famous monograph *Du délire de persécution*, in which he dissociated from the classifications of his predecessors that group of the insane whose symptoms are essentially those of the disease under consideration. A few years later Morel pointed out the existence of cases in which there occurred



in the course of the disease a transformation of the mental disposition, of the delusions, and of the hallucinations. Patients who were at one stage depressed, miserable, subject to delusions of persecution and disagreeable hallucinations, passed into a state of gaiety, with delusions of grandeur and agreeable hallucinations. In 1871 Foville described what he believed to be a hitherto unobserved form of insanity, which he called megalomania. He acknowledged the truth of Morel's observation regarding the transition in certain cases from persecution to ambition, but he included in his megalomania other clinical forms, in which grandiose delusions and delusions of persecution coexisted or alternated. The opinions of the two last named writers were greatly modified by Falret, to whom credit is due for elucidating still further the tangled skein of the problem of paranoia. It remained for Magnan, however, to give to the world in bold relief the clear description of systematised insanity which will always be associated with his name. In his monograph entitled *Le Délire chronique à évolution systématique*, the latest teaching of the French school, paranoia is divided into two great groups, namely, (1) Progressive systematised delusional insanity, in which the course of the disease is slowly progressive, and invariably commencing with depression and delusions of persecution, passes into ambition with expansive delusions; and (2) an affection, not necessarily progressive, characterised by irregular manifestations of systematised delusion either of a depressed or of an ambitious nature. The latter is termed by Magnan the systematised insanity of the degenerate. With few exceptions, English authors are in the position in which Prichard left the subject upwards of sixty years ago. The views of German psychiatrists will be best understood from the following references to the writings of one or two of their leading authorities. Schüle divides paranoia into two groups, *Originäre Verrücktheit* (delusional insanity of the degenerate) and *Chronischer Wahnsinn* (chronic systematised insanity). The latter comprehends persecution mania and ambitious delusional insanity or megalomania. Krafft Ebing classifies paranoia as follows:—(1) *Primäre Verrücktheit*, which he subdivides into (a) *Originäre Verrücktheit* (commencing in childhood), and (b) Paranoia tardiva, which includes the insanity of persecution and megalomania; (2) *Hallucinatorische Wahnsinn*, characterised by the excitation of the sensory centres in an exhausted brain. In this division are included a host of dissimilar conditions, such as acute alcoholism, forms of puerperal insanity, post febrile insanity, and certain mental affections found among prisoners. A majority of German psychiatrists have been influenced by Krafft Ebing's teaching, with the result that not only was the identity of paranoia in danger of being lost, but that considerable misunderstanding, due to a confusion of nosological terms, arose between German writers and their *confrères* in other countries. Fortunately, Kraepelin has recently raised his voice in protest, and in the last edition of his text-book has ably combated the inclusion of such absolutely different symptomatic forms as those last mentioned under the head of paranoia. His conception of paranoia is that of a systematised delusional insanity, commencing with persecution, but always accompanied by gradually developing delusions of grandeur. A second form, comprising a partially systematised insanity of the degenerate, he terms *Der Querulantenwahn*. To a corresponding class of the insane the French writers give the name *persécutés persécuteurs persécuteurs*. Both terms are expressive and descriptive of a group of patients who consider themselves wronged by society, and who are constantly demanding redress.

From the above résumé of the writings of various authors during the



past century, it will be seen that a continuous consensus of opinion has isolated a disease, the clinical nomenclature of which is diverse. To this disease many generic names have been given, but it would appear that paranoia must now for the sake of uniformity be accepted. Confusion as to clinical forms has necessarily arisen. The monomania of English writers is obsolete, and they themselves have done nothing to justify it. The paranoia of the Germans is in a state of chaos, out of which Kraepelin has done much to redeem it, but compared with the fulness of Magnan's descriptions, his presentation of clinical forms is meagre and unsatisfactory. There remain the *délire chronique* and the *délire des dégénérés* of the French school. In favour of this view two arguments may be submitted: First, the undoubted prevalence of the affection in France; and, second, the trustworthiness of the men who have devoted their lives to the study of the affection. The names of these men, some of which have been already mentioned, are among the most eminent of European psychiatrists. But the proof of the truth of the French teaching will only be found in the inner conviction which results from a clinical study of the disease as depicted by their writers.

Under the title borrowed from the Germans, the description of the affection which follows is on the lines of the French psychiatrists, and in accordance with their teaching it is divided into I. Progressive systematised insanity, and II. The systematised insanity of the degenerate.

#### I. PROGRESSIVE SYSTEMATISED INSANITY

*Definition.*—This disease is a chronic mental affection characterised by delusions of a fixed kind, and by hallucinations of hearing, of touch, and of pain. In the course of the affection there occurs a complete transformation of the personality of the patient and of the earlier symptoms of the insanity, accompanied by the appearance of delusions of an ambitious character, with corresponding hallucinations of hearing. Magnan divides the affections into four stages. The first period, or period of incubation, is characterised by illusion, insane interpretation, and mental anxiety. In the second period, or stage of persecution, the principal phenomena are delusions of persecution, hallucinations of hearing and of general sensibility. The third period, or stage of ambition, presents hallucinations of hearing of an agreeable character, along with delusions of grandeur. The fourth and last period is that of failing intellectual power or dementia.

*Ætiology.*—The disease manifests itself in adult life usually after thirty years of age, and in persons who had previously been of sound health and free from intellectual disorder. Hereditary predisposition does not play an important part in the production of the disease, although it exists in a large proportion of cases. The disease is more common in the female sex, and is more prevalent among married women who have led unhappy domestic lives. Prolonged disappointment, reverses of fortune, remorse, and jealousy are among the causes usually assigned. The physical causes cited as conducing to the malady comprise those influences which give rise to debility.

*Period of Invasion.*—The period of invasion is generally a slow and gradual one, the disorder manifesting itself at first indefinitely with malaise and uncomfortable sensations which forcibly attract the attention of the patient, especially when the sensations are of an intense nature. The appetite for food, the capacity for work and sleep are all diminished. The subjects of these disordered functions and sensations become suspicious, and imagine that there is something peculiar in their appearance which may be



visible to other people. From such ideas the transition is easy to the belief that they are stared at for some purpose, that they are despised or distrusted, or socially tabooed, until ultimately they lose self-control, and openly accuse people of annoying them. To these imaginations the patients add a review of their past life, persistently dwelling upon slights, insults, and oversights which they may have previously experienced, until they become convinced that they have long been the victims of systematic persecution. In some cases the symptoms of the invasion period are so vague as not to attract the attention of other people, or so short that the appearance of hallucinations of hearing which usher in the second stage may be mistaken for the commencement of the malady.

*Second Period.*—Hallucinations of hearing of a disagreeable nature are a constant symptom of the second stage. They begin vaguely as sounds or noises in the ears, and proceed steadily until isolated words are formed, and finally complete sentences and even long conversations are heard by the patient. In the majority of cases these hallucinations are unilateral, but in some cases bilateral. In the latter case it is not unusual for the patient to be cognisant of different voices in each ear. Hallucinations of hearing persist throughout the disease, but when the second stage is established other hallucinations commence to appear. First among these are hallucinations of taste and smell, upon which the patients found their delusions of poisoning, the most persistent delusions of the disease. Succeeding these come those of touch, pain, and general sensibility, under the influence of which the patients are often driven to retaliative measures against their supposed tormentors. Lastly, genital and sexual hallucinations make their appearance. These are usually of an intense and distressing character, absorbing the patient's whole attention, and disturbing his self-control more than any other kind of false perception. In men they may give rise to a belief in attempts at castration, emasculation, bleeding from the penis, venereal inoculation, or attempts at sodomy; in women, of pregnancy, obscene practices, rape, and the introduction of foreign bodies into the vagina.

The state of sensory hallucination is the most prominent and characteristic stage of the disease. The sufferings of the patient are often intense in this period, which may last from two to twenty years without any modification in the symptoms of the disease. It is important to observe, however, that all the symptoms are during this stage subject to remissions, in some cases to a slight extent, while in others there may be complete relief for periods varying from a few days to several months.

*Third Period.*—Period of exaltation and ambition. In proportion as the disease advances, and at the end of a time which varies with each case, ideas of grandeur are superadded to the existing ideas of persecution. The delusions of persecution do not at once pass away; in many cases they persist during the life of the patient, but they gradually lose their intensity, and may only remain in the mind as a disagreeable memory. Along with the delusions of ambition, hallucinations of hearing of a corresponding nature displace the previous hallucinations of the second stage, which are always disagreeable. The delusions of ambition are extremely various, and include those of pride, grandeur, wealth, and temporal and supernatural power. They are well defined and systematised, but in their expression there is manifested a tendency towards mysticism, with the formation of new and bizarre words, and a haziness bred of the difficulty of reconciling the life of phantasy with the true environment of which the patient is normally conscious.



*Fourth Period.*—Period of dementia. From the stage of ambitious delusion to that of dementia the progress is slight and gradual. The dementia is not equivalent to what is known as secondary or terminal dementia, but a mental weakness of a mild kind, a form of intellectual lowering in which, nevertheless, the patients retain their physical activity and their power of conversing in a rational manner on various subjects outside their delusions. The mysticism, to which reference has been made, markedly increases, and there is a tendency to wandering of the thoughts, and an absurd extravagance of expressing opinions in connection with the delusions. Some authorities, notably Falret and Rith, do not recognise the period of dementia.

*Prognosis.*—The prognosis is very unfavourable, and no treatment hitherto discovered has any influence in modifying the invariable and inevitable course of the malady. Most of the patients require to be confined, and their treatment in asylums is purely moral and symptomatic.

## II. SYSTEMATISED INSANITY IN THE DEGENERATE

The description already given of progressive systematised insanity is sufficient to indicate the broad lines of the malady as it is commonly met with. The symptoms do not differ from those observed in the degenerate, but the course of the affection in the latter is different. One important distinction is the rapidity with which insanity in the degenerate may become systematised. Slowly and under the prolonged influence of hallucinations the delusions in the chronic form develop, but in the degenerate they may burst forth, fully formed, without the accompaniment of hallucinations. The manifestation of systematised insanity in the degenerate is also more irregular and varied. The onset of the insanity may be slow and insidious, or quick and explosive. In the first instance the patient is usually a recluse, and reasons out his own symptoms step by step, or he consults books of every description, from which he culls irrelevant passages, which he believes to have a bearing on his own case. The affection may present itself in any of the following forms:—

(1) The commencement may be ambition, with delusions of exaltation. A patient who has previously been reticent and shy may all at once be convinced that he is born to great things and with a special destiny. Such people deny their parentage, and assign to themselves exalted origin. Yet these insane ideas appear to have little root, and the belief in their reality does not seriously affect the real life of the subject. Gradually delusions of persecution come to be added to the ideas of grandeur. The patient imagines that his name or his exalted position renders him an object of suspicion and hatred to others, who, by plotting and persecution, wish to deprive him of his rights. From this point a regular systematised insanity of persecution is developed.

(2) Delusions of persecution may manifest themselves first of all. These appear to originate very much after the manner of imperative ideas, and are usually systematised without the aid of hallucinations, which come on, as a rule, later in the course of the disease. They in their turn may be followed by delusions of pride and grandeur and an ambitious insanity.

(3) The two forms (ambition and persecution) may burst out simultaneously, progress side by side, and succeed each other alternately, causing periods of depression and exaltation.

(4) The manifestations of the systematised delusional insanity may be

preceded by, interrupted by, or terminated by attacks of acute insanity, such as mania, melancholia, or stupor.

(5) The character of the insanity varies according to the intellectual development of the patient. When the patient is of limited intelligence the delusions are usually ridiculous and absurd, but when the intelligence is of a higher order they correspond more to the tone and definiteness of those of a case of true systematised insanity. But, as has been indicated, the character of the delusions of the degenerate is feeble and lacking in conviction and reality. This feature and the variability of the symptoms depends upon the weakness and exceedingly unstable mental constitution of the patients.

(6) The terminations of the affection are also numerous. Sometimes the cases pass through a whole series of changes—ambition, persecution, with recurrent maniacal melancholic or stuporose attacks, and ultimately recover. In others the character of the symptoms frequently alters, but the end is dementia. Others, again, pass through a metamorphosis from ambition to persecution, or *vice versa*, and remain in the latter phase until recovery or during life.

*Prognosis.*—It is unnecessary to state that in such a hydra-headed group of insanities prognosis must be a matter of great difficulty. Generally speaking, it is in the majority of cases unfavourable. The more acute the symptoms the more rapidly the insanity changes, and the more frequent is the admixture of acute general conditions, such as mania and melancholia, the more favourable is the prospect. Conversely, the more chronic the symptoms are, the more unchangeable the insanity, and the more confirmed the mental condition which underlies all the symptoms, the more unfavourable the prognosis.

The term “secondary paranoia” has been applied by some writers to the delusional states met with in certain forms of melancholia, especially anxious and hypochondriacal melancholia, and to the delusion met with in some forms of terminal dementia. In the former the delusions bear little or no resemblance to those of paranoia, and in the latter they are too indefinite and too loosely systematised to be entered into a separate classification.

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## Paraplegia. See PARALYSIS.

## Parasites.

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ALTHOUGH parasites belong both to the animal and vegetable kingdoms, only the animal parasites are considered in the following article.

Parasitic animals are those which, in order to obtain their nourishment, live within, or upon another living organism, termed their host. True parasites nourish themselves on the living material (blood, lymph, etc.) of their host; saprophytic parasites obtain their nourishment from lifeless material, such as that within the alimentary canal, or that on the outer surface of the host. Many parasites are pathogenetic; but though some of the saprophytic parasites do not harm their host, it is doubtful whether any animal parasites are "symbiotic," or, in other words, whether any are beneficial to their host.

Some parasites, *Trichina spiralis* and *Fasciola hepatica*, for example, are found in different species of animals, whilst other parasites are limited to a single species. Thus the adult *Tænia solium*, *Strongyloides intestinalis*, and *Oxyuris vermicularis* are parasitic only in man, and *Tænia serrata* is found solely in the dog. The present article deals only with the parasites of man, exception being made, however, of a few species parasitic in the lower animals, which are referred to on account of their close relationship to species parasitic in the human subject, or because a knowledge of such parasites is of importance in a study of the parasites of man.

The anatomical situation of a parasite is known as its habitat, animal parasites being designated Ectozoa or Entozoa according as they live upon or within their host. Each parasitic species has what may be termed a normal habitat. That of the adult Cestodes, for example, is the intestinal canal, that of *Sarcoptes scabiei* is the skin, that of *Echinococcus polymorphus* certain internal organs. In some instances, however, parasites are found in abnormal situations; *Ascarides*, for example, may be found in the respiratory passages.

The parasitism of some animals is transitory, that of others is permanent.

The mode of reproduction within the body of the host is very varied. Some parasites, for instance the protozoa, produce successive generations within the host; others, for instance the worms, when within the host merely attain sexual maturity, and give origin to a second generation, which does not forthwith become sexually mature in the same host. In some

instances this second generation becomes mature, however, after re-entering the same host, or after gaining entrance to another host of the same species. In other instances two specifically different hosts are necessary for completion of the life cycle of the parasite, which is immature in the one and sexually mature in the other host. In the latter instance, then, a change or alternation of host is requisite for the developmental cycle of the parasite, and the animal harbouring the sexually mature parasite is the *definitive* host, that harbouring the immature parasite is the *intermediate* host.

There are various modes in which man is infected. The ingestion of eggs or immature forms of parasites together with water or vegetables is one of the most common. By the ingestion of eggs man is infected with *Cysticercus cellulosæ*, *Echinococcus polymorphus*, *Ascaris lumbricoides*, *Oxyuris vermicularis*, etc.; by the ingestion of immature species he is infected with *Uncinaria duodenalis*, *Strongyloides intestinalis*, and Trematodes. Again, infection may be brought about by ingestion of the immature parasites in an intermediate host. It is thus that man is infected with the adult Cestodes and with *Trichina spiralis*. And, lastly, parasites may be transmitted by the direct agency of the second host, as is the case with Filariidæ and Hæmosporidia.

Animal parasites cause disease in a variety of ways, of which the most important are:—1. By mechanical injury; for instance, when an *Echinococcus* growing within the liver exerts pressure on neighbouring structures, or when *Ascarides* wander into the bile-ducts, or when *Trichinæ*, *Ascarides*, or other worms penetrate the intestinal wall. 2. By the presence of the parasite exciting inflammatory reaction in the tissues (larval *Trichinæ*, *Cysticerci*, etc.) 3. By withdrawal of nutrient material, as when there are many large intestinal worms. 4. By removal of blood, e.g. by *Uncinaria duodenalis* and others. 5. By production of poisonous substances, e.g. malarial protozoa, Cestodes, *Uncinaria duodenalis*, *Trichocephalus trichiurus*, etc. (v. Linstow, 1896, Peiper, 1897, and many other authorities).

The various substances which resemble, and may be mistaken for parasites are termed pseudoparasites.

The animal parasites may be considered under the three headings:—

A. Protozoa, B. Parasitic worms (Helminths), C. Arthropods.

## PROTOZOA

The protozoa are unicellular animals, the simplest of all animal organisms. Their general form and structure, the modes of movement, nutrition, and multiplication have been considered in vol. viii. pp. 72, 73. Their classification into five classes is the most satisfactory. Four of these five classes have to be considered, namely:—

- |  |                       |
|--|-----------------------|
| I. Movement effected by pseudopodia . . . . .  | <i>Rhizopoda</i> .    |
| II. Movement effected by flagellæ . . . . .  | <i>Mastigophora</i> . |
| III. Movement variously effected, usually impaired. Multiplication by numerous encapsuled reproductive bodies (spores) . . . . . | <i>Sporozoa</i> .     |
| IV. Cilia present throughout life, nutrition by osmosis or cytostoma . . . . .   | <i>Ciliata</i> .      |

RHIZOPODA; Syn.: *Sarcodina*.—The cell protoplasm is either simple or differentiated into ecto- and entoplasm. Movement and nutrition occur by means of pseudopodia, reproduction is by division. Of the five orders included in this class only two, *Amœbina* and *Mycetozoa*, are parasitic. But



the latter are parasitic only on plants, hence only the parasites belonging to the order *Amœbina* have to be considered.

Genus *Amœba*.—Many organisms of this genus inhabit fresh or salt water, others are parasitic. Within the last few years attempts, but hitherto without success, have been made to isolate and cultivate amœbæ on artificial media, in some instances by providing bacteria as the source of nourishment (Ogata, 1893; Celli and Fiocca, 1896; Schardinger, 1896; etc.).



FIG. 1.—*Amœba coli*. (Braun after Kovacs.)

Doflein (1901) considers that the cultures obtained have usually been *Mycetozoa* and not true amœbæ.

*Amœba coli* (Loesch, 1875) is 10-50  $\mu$  in diameter, rounded or irregular, uninuclear, has coarsely granular entoplasm with several non-contractile vacuoles, and often contains foreign particles; pseudopodia are short and finger-like. Multiplication is by division whilst still motile or after becoming encysted. The encysted amœbæ possess a much greater power of resistance than does the ordinary amœba, and it is possible that infection of the host occurs by the ingestion of the former. *A. coli* has been found most frequently in the fæces and large intestine of man, though also in water and soil polluted by dejecta. The parasite has been observed in the stools of healthy persons and of those suffering from dysentery, typhoid fever, cholera, etc., and also in the pus of tropical hepatic abscesses. The chief interest of *A. coli* lies in its association with dysentery, for although a direct etiological connection of the parasite with this disease has hitherto not been proven, it is generally held that in those types of the disease designated "amœbic dysentery," *A. coli* plays a part together with one or more species of pathogenetic bacteria in the causation of the disease. It is suggested, but on grounds which appear quite indecisive, that there are at least two distinct species of intestinal amœbæ—the one, *A. dysenteriae*, found only in dysentery, and differing from the ordinary saprophytic *A. coli* in being smaller and more finely granular.

Further details regarding *A. coli* will be found in the articles on dysentery, and on tropical abscess of the liver in vol. vii.

Amœbæ, possibly identical with *A. coli*, and of doubtful pathogenicity, have been observed in the urine of patients suffering from hæmaturia, within cysts in the mucosa of the urinary bladder, and in a sequestrum from the inferior maxilla. Saprophytic amœbæ are also found in the intestine of several species of animals, but it is doubtful whether *A. parasitica*, described by von Lendenfeld (1885) as causing a fatal skin disease in lambs in Australia, is a protozoon.

The rhizopods found by Leyden and Schaudinn (1896) in ascitic fluid resemble amœbæ in many respects.

MASTIGOPHORA.—All the parasitic organisms are contained in the subclass of the Flagellata. These all possess flagellæ; nutrition is maintained in a variety of ways; multiplication is by fission, either when in a free or resting stage. There is frequently a sexual multiplication also. Many flagellates are parasitic in insects. Those which concern us here are all included in one or other of the following genera—*Cercomonas*, *Trypanosoma*, *Trichomonas*, or *Lamblia*.

The organisms of the genus *Cercomonas* are ovoid or spherical, small and colourless, and have one flagellum anteriorly. They have on many occasions been detected in the sputum, lungs, and pleuritic exudations both of man and the lower animals, and are no doubt merely saprophytic.



*Trypanosomata* are mostly of an elongated and spiral form, uninuclear, with usually only one flagellum anteriorly and actively motile. Their development is as yet imperfectly understood.

*Trypanosoma Lewisi* (Kent, 1882), 8-20  $\mu$  long by 1-2  $\mu$  broad, was found by Lewis (1879) in the blood of rats. In them it is usually saprophytic, and is possibly identical with similar organisms in the blood of certain fishes. It is probably specifically distinct from

*Trypanosoma Brucei* (Plimmer and Bradford, 1899).—This resembles *T. Lewisi* morphologically, and is about 10-12  $\mu$  long. The posterior extremity is obtuse. It is found in the blood of the ox, horse, mule, camel, etc., and can be transmitted to the dog and rat. It is as shown by Bruce (1894, 1897) the cause of tsetse fly disease, or nagana, in Central, East, and South Africa, the disease being transmitted by the tsetse fly, which infects the animals by its bite. The disease is not transmissible to man.

*Trypanosoma Evansi*, first recorded by Evans in 1880, is 20-30  $\mu$  long by 1-2  $\mu$  broad, and has the posterior extremity pointed. It is parasitic in the blood of the horse, camel, goat, and other animal species in India and the Dutch East Indies, causing chiefly in the horse and mule the disease termed surra, of which irregular fever and anæmia are prominent symptoms. The disease is possibly inoculated by the bite of *Tabanus tropicus*. E. R. Rost (1901) finds that the inoculation of surra blood into goats is followed by a fever of short duration, but that the subsequent injection of their blood serum into mules and ponies is the reverse of beneficial. It is not yet decided whether surra and tsetse fly disease are the same or different diseases. The clinical signs of both are very similar, but the tsetse fly is not known to exist in India.

*Trypanosoma of dourine* was first described by Rouget (1896) as being 18-26  $\mu$  long by 2-2.5  $\mu$  broad. It is found in the blood of mares and stallions, causing the disease known as *dourine* in France and as *die Beschälseuche* in Germany. The disease is transmitted by coitus, and is now regarded as specifically distinct from surra and tsetse fly disease.

Other organisms belonging to the same family as the trypanosomata are found in the intestine of the housefly and of certain birds, and in the blood of fishes, frogs, etc., but it is doubtful whether any are parasitic in man.

Of the different species belonging to the genus *Trichomonas* only two are found in the human subject. They are I. *Trichomonas vaginalis* (Donné, 1837), spindle or pear-shaped, usually about 17  $\mu$  long, the posterior end pointed, the anterior bearing three flagellæ. It is a common saprophyte in acid vaginal mucus, and has also been found in the urethra and urine from both sexes. II. *Trichomonas hominis* (Davaine, 1854); Syn.: *Cercomonas hominis*; etc., is pyriform, 4-10  $\mu$  long, with three flagellæ anteriorly. It is found in the healthy intestine and in the fæces of persons suffering from diarrhoea, but is probably a mere saprophyte (Fig. 2).

*Lambliæ intestinalis* (Lambl, 1859).—It is pear or carrot shaped, 10-16  $\mu$  long, shows a suckorial depression anteriorly, has two long flagellæ attached to the pointed posterior extremity, and three pairs of flagellæ to the anterior half of the body. It inhabits the upper half of the small intestine of man and many of the lower animals, and is also found in the fæces when the host suffers from diarrhoea. Like *T. hominis* it is saprophytic.

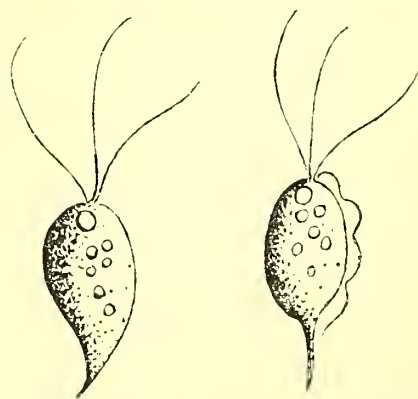


FIG. 2.—*Trichomonas hominis*. (After Grassi.)



SPOROZOA are all parasitic. At one stage in their life-cycle they all multiply by the splitting off of encapsuled bodies, which are often termed "spores." Owing to the rapid growth of knowledge in regard to the sporozoa, there is at present no system of classification generally accepted and no nomenclature universally adhered to, although any species should be referred to by its original designation, whilst synonyms subsequently introduced are discarded. The following classification is that often adopted at present:—

I. Subclass *Telosporidia*, containing the following orders: *Coccidiida*, *Hæmosporidia*, *Gregarinida*, *Amæbosporidia*.

II. Subclass *Neosporidia*, with three orders: *Myxosporidia*, *Microsporidia*, and *Sarcosporidia*.

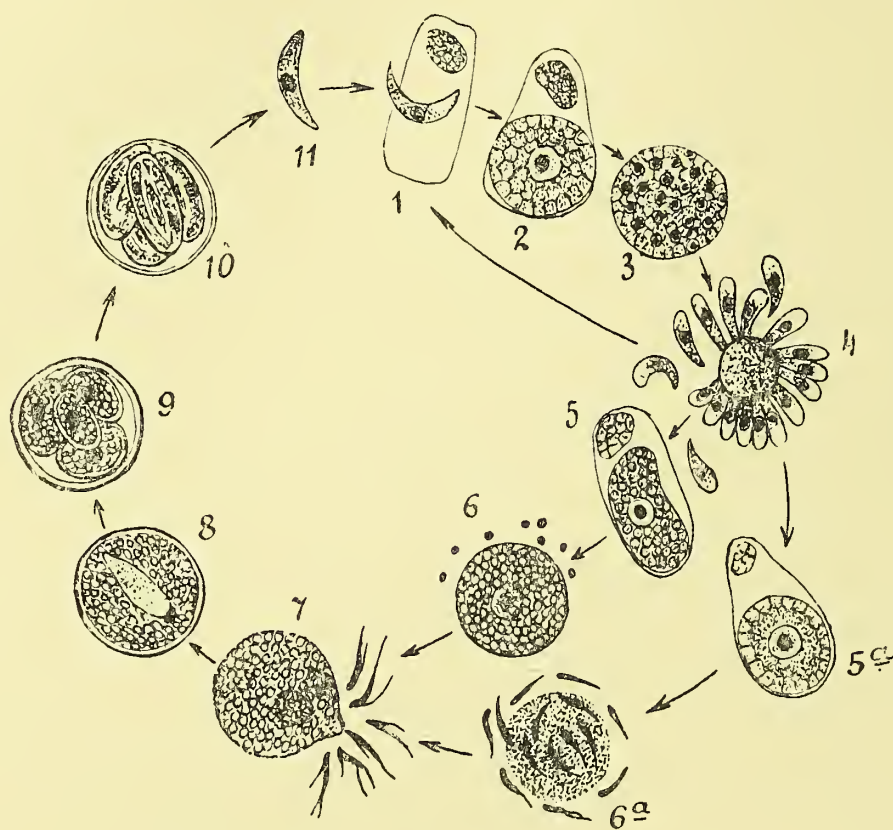


FIG. 3.—Diagram of the developmental cycle of *Coccidium Schubergeri*. (Lühe, after Schaudinn.) 1. Sporozoite (or merozoite) penetrating into an epithelial cell; 2. Fully developed schizont; 3. Nuclear proliferation preparatory to; 4. Formation of merozoites; 5 and 5a. Sexual elements (5, macrogamete; 5a, microgametocyte); 6 and 6a. Ripe sexual elements (6a, microgametocyte forming microgametes); 7. Fertilisation; 8. Young oocyst; 9. Sporoblast formation in the oocyst; 10. Formation of sporozoites within sporoblasts transformed into sporocysts; 11. Sporozoite.

*Telosporidia*.—In all the sporozoa belonging to this subclass, "spore" formation occurs only when the growth and existence of the individual organism is about to terminate. The adult telosporidia are all uninuclear, and the life-cycle of each commences by the invasion of a cell of the host.

I. *Coccidiida* are parasitic in epithelial cells chiefly of the intestine or abdominal viscera, rarely in connective tissues. They are found in arthropods, amphibia, fishes, birds, and mammals. Rabbits are frequently the host, and man harbours certain species.

The life-cycle of coccidiida closely resembles that of hæmosporidia. In fact, with a single exception, there are two alternating generations with two modes of multiplication—(1) a sexual multiplication by "spore" formation (sporogony); and (2) an asexual by fission (schizogony), both occurring without a change of host. The life-cycle of *Coccidium cuniculi* will serve as an example. (i.) *Sporogony*.—In the fæces of infected rabbits there are oval or spherical encapsuled parasites termed oocysts. Outside the body of

the host, the sporont, or uninuclear content of an oocyst, divides into usually four cells (sporoblasts), and each sporoblast acquiring a highly resistant membrane is now termed a "spore" or sporocyst. Each sporocyst then divides into two uninuclear crescentic bodies (sporozoites), and if the sporocysts be ingested by a suitable host the sporozoites penetrate into epithelial cells—for example, those of the bile-ducts. (ii.) *Schizogony*.—A sporozoite having entered a cell alters its form, enlarges, and is termed a schizont, which by multiple division gives origin to many merozoites, some of which again enter epithelial cells and develop into schizonts. Other merozoites develop into either female or male elements, termed respectively macrogametes and microgametocytes, each of the latter giving origin to many microgametes. A macrogamete having been fertilised by a microgamete a young oocyst is produced. The process of "spore" formation results in the infection of many epithelial cells, that known as schizogony is concerned also with the infection of other hosts.

*Coccidium cuniculi* (Rivolta, 1878); Syn.: *C. oviforme* (Leuckart, 1879).—

The oocyst (stage 8, Fig. 3) is oval, and measures 33-40  $\mu$  long by 14 to 28  $\mu$  broad. Most of the coccidia seen in the tissues of the host are at this stage of development. The presence of these sporozoa causes the disease designated coccidiosis; the term psorospermiosis is not so precise and should be discontinued. The disease is most frequent in young rabbits,—Délepine (1890) found lesions visible to the naked eye in 92 per cent,—and is rare in man, the horse, ox, pig, and goat. In all cases, infection doubtless occurs by the ingestion of food contaminated by the faeces of an affected rabbit. In the rabbit the disease is either acute or chronic, and

either terminates fatally or in recovery. The liver is found enlarged, and both on its surface and on section presents numerous grayish white nodules or cysts—dilated intrahepatic bile-ducts—varying in size from a hemp-seed to a hazel-nut, and each containing a thick yellowish or creamy débris. On microscopic examination the walls of these dilated bile-ducts are seen to be thickened by much fibrous tissue, and present papilliferous outgrowths towards the lumen which is more or less filled with oocysts. These must not be mistaken for ova of trematodes which they resemble in shape. Many of the lining epithelial cells contain schizonts, others are desquamated. Coccidiosis in the liver of the human subject is a rare disease. Cases have been recorded by Gubler (1858), Silcock (1890), and others. The liver has shown essentially the same changes as are observed in the livers of infected rabbits. Podwysoski (1889), however, stated that the coccidia also inhabit

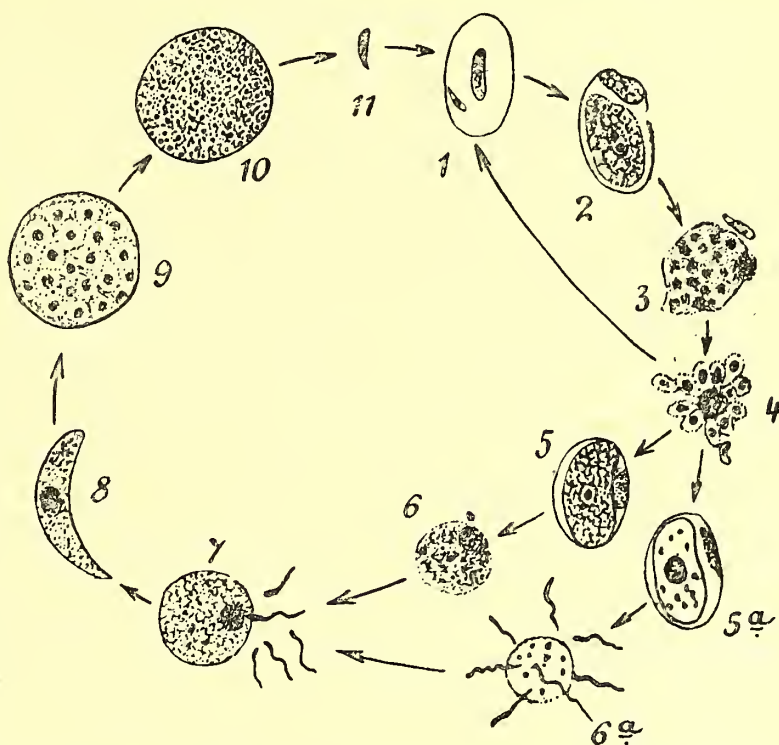


FIG. 4.—Diagram of the developmental cycle of *Harmoproteus* (Lühe after Schaudinn.) 1. Sporozoite (or merozoite) within a red blood corpuscle; 2. Fully developed schizont; 3. Nuclear proliferation; 4. Formation of merozoites; 5 and 5a. Sexual elements (5, macrogamete; 5a, microgametocyte); 6 and 6a. Ripe sexual elements (6a, microgametocyte forming microgametes); 7. Fertilisation; 8. Ookinete; 9. Sporoblast formation in the oocyst; 10. Formation of sporozoites; 11. Sporozoite.



the liver cells, and are to be found widely scattered through the organ. There is often some diffuse cirrhosis, and the patients have suffered from anæmia, jaundice, and emaciation.

In one instance *C. cuniculi* is claimed to have caused the development in the brain of a nodule of bone as large as a pea (J. J. Thomas, 1899).

Lastly, the coccidia associated with the disease termed *die rothe Ruhr*, a hæmorrhagic diarrhœa of oxen in Switzerland, are regarded as identical with *C. cuniculi* (Guillebeau, 1894; etc.).

*Coccidium hominis* (Rivolta, 1878); Syn.: *C. perforans* (Leuckart, 1879), is possibly identical with *C. cuniculi*. It is parasitic in the intestine chiefly of young rabbits, causing enteritis which is often fatal. The coccidium is found in the intestinal epithelium, sometimes also in the submucous tissue and mesenteric glands. Two cases of coccidiosis of the intestine in the human subject were recorded by Eimer in Berlin (1870).

*Coccidium bigeminum* (Stiles, 1892) measures 8-15  $\mu$  long by 6-10  $\mu$  broad, and is found in the substance of the intestinal villi of the cat, dog, and other mammals. The coccidia found in the human subject by Kjelberg (1860), and Railliet and Lucet (1893), are possibly examples of *C. bigeminum*.

*Coccidium avium* (Silvestrini and Rivolta, 1873); Syn.: *C. tenellum* (Railliet and Lucet, 1891).—It has been detected in fowls and pheasants, and regarded as the cause of an enteritis at times fatal; it is also claimed as the cause of diphtheritic inflammation of the mouth, pharynx, and larynx of fowls and other birds (M'Fadyean, 1894; and others).

Structures, regarded as coccidia by those who detected them, have been seen in one case in a pleural exudation (Künstler and Pitres, 1884), in other instances in the kidney and ureters, and, lastly, in the pearly growths of molluscum contagiosum.

II. *Hæmosporidia* are telosporidia in which schizogony takes place in the blood of vertebrates, and sporogony occurs in arthropods. The vertebrate animal is the intermediate host, the arthropod the definitive host. In many respects our knowledge of the hæmosporidia is still very incomplete, and largely on this account there is no general agreement as to their classification and nomenclature.

In view of these facts, and remembering that a classification which to-day appears correct will probably be antiquated in a few months, it is manifestly inadvisable to give any classification here. One of the most recent will be found in the monograph of Neveu-Lemaire, *Les hématozoaires du paludisme* (1901).

The life-cycle has been most fully worked out in certain hæmosporidia, for example *Plasmodium malariae*, which undergo sporogony in mosquitos. The cycle has been already described in detail in the article on malaria. All that is therefore necessary is to give a diagrammatic representation of the life-cycle (Fig. 4) in order to illustrate the close analogy between that of the hæmosporidia and that of the coccidiida.

*Hæmosporidia of batrachians and reptiles*.—There are a number of different species. Frogs, turtles, and lizards are the chief hosts. At certain stages of development the parasites are situated in the red blood corpuscles; at other stages sickle-shaped and elongated bodies are free in the blood serum. The life-cycle is imperfectly understood, but Schaudinn (1899) suggests that these hæmosporidia require no alternation of host in order to complete their development. There is but little hæmolysis caused by most of these hæmosporidia.

*Hæmosporidia of birds* have been detected in pigeons, owls, crows,



sparrows, starlings, and other birds. The parasites are usually grouped into two or three species:—

1. *Halteridium Danilewskii* (Labbé); Syn.: *Hæmamæba Danilewskii* (Grassi and Feletti); etc., is a common parasite in many parts of the world, but not markedly pathogenetic. The sporozoites penetrate into the red blood corpuscles and develop into pigmented, round, elongated, or constricted bodies. The asexual cycle in the avian host is completed in seven or eight days. Sexual elements have been found in the blood plasma, but sexual multiplication occurs within species of *Culex*.

2. *Hæmoproteus Danilewskii* (Kruse); Syn.: *Proteosoma Grassii* (Labbé); etc., is common in all countries, and more highly pathogenetic than the last-mentioned species, causing avian malaria. The intracorpuseular parasites are often spherical, and usually pigmented with melanin. The asexual formation of merozoites is readily observed in the blood of the host. Schizogony is completed in four or five days, sporogony in the middle intestine of species of *Culex* (*C. pipiens*, *C. fatigans*) is completed in about nine days. The development in the mosquito was first studied in the case of this parasite by Ross (1898), who was likewise the first to demonstrate the fact that birds acquire malaria solely by the bite of infected mosquitos. Infection with this parasite causes among other signs fever—which is not influenced by the administration of quinine—and anæmia, and not unfrequently terminates fatally.

*Hæmosporidia of the human subject.*—1. *Malaria*. The hæmosporidia are described in the article on malaria. 2. *Blackwater fever* is regarded as due either to a “malarial parasite,” or more probably to a distinct, though as yet unknown species of the hæmosporidia (*vide* “Blackwater”).

*Sporozoa, probably Hæmosporidia, of other Mammals.*—(a) *The Ox*. Bovine malaria is a disease which occurs in many countries. In Mexico, Carolina, and other adjacent parts, the disease is termed Texas fever (Syn.: *Redwater fever*). The description of the hæmosporidium, *Pyrosoma bigeminum*, causing Texas fever is largely based on the researches of Smith and Kilborne (1891-93). The intracorpuseular parasite at an early stage is a small ( $2.5-4\ \mu \times 1.5-2\ \mu$ ) amœboid body of irregular form and at a somewhat later stage often appears in the form of two pears joined by their stalks, whence the name. The asexual development of the intracorpuseular parasite is not yet fully investigated, but it may be mentioned that Doflein (1901) considers that the pyriform bodies correspond to the crescents of the plasmodia of human malaria. The disease is transmissible to healthy oxen by the injection of blood from an animal suffering from Texas fever, just as birds may be infected with avian malaria by the injection of the blood of other birds acting as the hosts of *Halteridium* or *Hæmoproteus*, or as malaria of the human subject has been transmitted to healthy persons from those affected with the disease. Bovine malaria is, however, ordinarily transmitted by a species of tick (*Boophilus bovis*), within which, however, the development of the parasite has still to be investigated. The female tick having been fertilised, and having sucked the blood of the affected ox, and thus been infected with *Pyrosoma bigeminum*, falls from the animal on to the pastures. A few days later about 2000 eggs are laid, from which in about two to six weeks the daughter ticks escape. It is by them that other oxen are infected. The disease appears in an acute or chronic form. In the former case the chief signs are pyrexia, hæmoglobinuria, and severe anæmia, whilst the chief pathological alterations observed post-mortem are those referable to the anæmia, and also acute enlargement of the spleen, con-



gestion of the liver, kidneys, stomach, and intestines. Diseases closely allied to, if indeed not identical with Texas fever, and in every instance propagated by ticks, have been recorded by Babes (1888-89, etc.) in Roumania, by Piana (1889-90) in Italy, by Celli and Santori (1897) in the Roman Campagna, by Jackschath (1901) in Pomerania, and by others in Finland, Sardinia, Portugal, South Africa, and East Africa; whilst in other parts of the world many cases of the condition termed "Hæmoglobinuria of the ox," or "Red water," are probably due to hæmosporidia. Many experiments have of late been undertaken with the object of rendering oxen artificially immune to Texas fever. To confer such immunity the animal must have been inoculated with a mild form of the disease, either by the use on two occasions of 2-5 cc. of blood of a convalescent animal, or infection may be carried out by means of *Boophilus bovis*. The resulting fever is mild, lasts about seven days, and there is, it is claimed, subsequent specific immunity. This assumption should, however, be accepted with some reservation.

(b) *Other Animals*.—The disease known as *Cârceag* which affects sheep in the delta of the Danube, has been referred to hæmosporidia by Babes (1888, etc.) and Starcovici (1893). A similar disease of sheep in the vicinity of Padua, described by Bonome (1894), and near Constantinople (Laveran and Nicolle, 1900), is regarded as due to intracorpuseular sporozoa. Hæmosporidia have also been found in dogs by Leblanc, Marchoud (1900), and others; in monkeys Koch found hæmosporidia closely similar to those causing human malaria; in bats Dionisi found two forms which resembled respectively *Plasmodium malarie* and *Plasmodium præcox* of man; and, lastly, though the subject requires investigation, there are the hæmosporidia recorded by Rickmann (1900) and others in South African horse sickness.

III. *Gregarinida* may be dismissed briefly, having never been observed in a vertebrate host, and none being pathogenetic.

IV. *Amæbosporidia* are parasitic in certain small beetles.

*Neosporidia*.—These sporozoa form "spores," whilst the growth and existence of the individual organism continues. The three orders included in this subclass are of importance chiefly from the standpoint of comparative pathology and biology. 1. *Myxosporidia* are parasitic chiefly in fishes, also in reptiles and amphibia. 2. Of *Microsporidia*, one species causes pébrine disease in silkworms. 3. *Sarcosporidia*. These are parasitic chiefly in the striated muscles of mammals, but also in birds and reptiles. The size of different species varies—often a few millimetres in length; they are usually cylindrical, and have a thick encapsuling membrane, the cavity within being divided up by protoplasmic partitions which separate the "pansporoblasts" one from another. Within the pansporoblasts sickle-shaped sporozoites are developed. Sarcosporidia are of little or no pathogenicity. Only one, or at most two definite cases have been recorded in man. Sarcosporidia are, however, common in the lower animals:—

*Sarcocystis Miescheri*; Syn.: *Rainey's tubes*, measure up to 2-3 mm. long, and are of common occurrence in the muscles of swine. The detection of the sickle-shaped sporozoites would serve in a doubtful case to differentiate this sporozoon from an encapsuled trichina.

*Balbiania gigantea* may attain the size of a small hazel-nut. It is found in the sheep and pig, chiefly in the œsophageal wall, tongue, and larynx.

*Doubtful sporozoa*.—*Coccidioides immitis* is probably one of the sporozoa, but is not a coccidium. It is found in the subcutaneous tissue of man,



causing lesions which resemble those of lupus or leprosy. Cases have been recorded by Wernicke (1892), Rixford and Gilchrist (1897), and A. Posadas, (1898).

CILIATA; Syn.: *Infusoria*, are characterised by the whole ectoplasm being covered with cilia. Three species have been found in the human intestine: (1) *Nyctotherus faba* (Schaudinn, 1899); found in one patient and probably saprophytic. (2) *Balantidium minutum* (Schaudinn, 1899); found twice in Berlin, probably saprophytic. (3) *Balantidium coli* (Malmsten, 1857) is egg-shaped, 40-110  $\mu$  long, and of somewhat complex structure. It is commonly regarded as a saprophyte, whether the intestine be healthy or the seat of inflammation. Two cases have, however, recently been recorded (Strong and Musgrave, 1901; Solowjew, 1901) in which the parasite apparently caused acute and fatal ulcerative enteritis in the large intestine. In Solowjew's case *Balantidium coli* was found in all the coats of the intestine, which presented marked signs of a vital reaction to the parasite. In the case recorded by Strong and Musgrave, the parasite was found between the muscular layers, and there were numerous eosinophile leucocytes especially in the submucosa.

*Doubtful Protozoa*.—As is well known there is an extensive literature on certain structures which are often regarded as pathogenetic protozoa. Examples are furnished by the "protozoa" of syphilis, measles, variola, and other infective diseases, by the "hæmamœbæ" of leucocythæmia and the "protozoa" of malignant tumours. In almost every instance proof is still lacking that these structures are really protozoa. They are all probably artefacts, cell inclusions or accidental structures of no etiological significance, and in regard to the malignant tumours, it is instructive to remember that zoologists are unable to accept any one of the so-called carcinoma parasites as a protozoon.

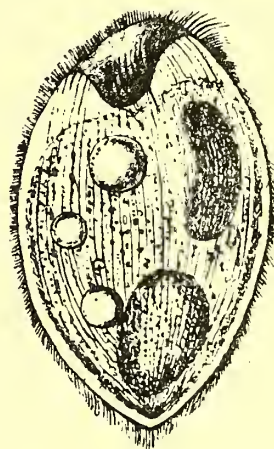


FIG. 5. — *Balantidium coli*. (After Leuckart.)

LITERATURE.—The literature on protozoa is very extensive. Among the most important of recent monographs are:—L. PFEIFFER, *Die Protozoen als Krankheitserreger*, 1891; *Ibid. Nachträge*, 1895; and F. DOFLEIN, *Die Protozoen als Parasiten und Krankheitserreger*, 1901. References to all recent literature are contained in *Baumgarten's Jahresh. u. . . d. path. Mikroorganismen*.

### HELMINTHS—*The Parasitic Worms.*

(A) PLATHELMINTHS ( $\pi\lambda\alpha\tau\acute{\iota}\varsigma$ , flat, wide), *platodes* or flat worms, have a flattened body. Their respiration is cutaneous, and there are usually organs of attachment, suckers or hooks. The chief mode of multiplication is a sexual one, and each animal is usually hermaphrodite. In some there is also an asexual multiplication by fission or budding. Some have an alimentary canal without an anus, others have no such canal. All the parasitic species are included in the two orders: Trematodes and Cestodes. They are either entozoa or ectozoa, but in the case of man, as well as of all the higher animals, they are all entozoa.

I. *Trematodes*, or flukes, are generally small plathelminths, of a leaf-shaped form, and are unsegmented. The alimentary canal has one opening, the mouth, at the anterior end of the body; the genital apertures and the suckers are situated on the ventral aspect of the body. The body is enclosed in a cuticular membrane which, in some species, bears numerous small spines, a parenchyma lies between this membrane and the wall of the



intestine and the other organs; there are muscular fibres in various parts, and these constitute also the chief elements of the suckers. In the entoparasitic trematodes there are never more than one or two suckers; the one is always at the ventral aspect of the somewhat pointed anterior extremity of the body, and, as it surrounds the mouth, it is termed the oral sucker. If, as is usually the case, there is a second sucker, it is situated on the ventral surface at a variable distance behind the former. The mouth is continued into a pharynx, an œsophagus, and an intestine which bifurcates into two cæca, or limbs, which pass backwards to near the posterior end of the body and end blindly. Most of the reproductive organs are situated between the two limbs of the intestine, and nearly all the trematodes are hermaphrodite. The eggs are formed in the uterus, and remain there for a considerable period of time, during which they ripen. When ripe they are of a brownish or yellow colour, and have, as a rule, an operculum. The eggs are eventually passed from the host by way of the intestine or urinary passages. The embryo or *Miracidium* then escapes from the egg, and its subsequent development varies in different species. In some, for instance, *Fasciola hepatica*, there are between the *Miracidium* and the adult worm two generations in a special intermediate host, and there is thus an alternation both of generations and of hosts.

With the exception of *Monostoma lentis*, which has only one oral sucker, and was found once within the lens of a woman, all the trematodes of man belong to one or other of the three families: Fasciolidæ, Amphistomidæ, Schistosomidæ.

*Fasciolidæ*.—There are two suckers, the posterior one being situated in the median line of the ventral surface, but not at the posterior extremity of the body. The parasites found in the human subject are all included in the four genera: *Fasciola*, *Dicrocoelium*, *Opisthorchis*, *Mesogonimus*.

*Fasciola hepatica* (Linné, 1758); Syn.: *Distomum hepaticum* (Retzius, 1786).—The body is 20-32 mm. long,



FIG. 6.—Eggs of *Fasciola hepatica*. (After Mosler and Peiper.)

8-13 mm. broad, flattened, oval or leaf-shaped, more pointed posteriorly than anteriorly, and of a pale brownish yellow colour. The oral sucker is small and circular, and 3 mm. behind it is the larger ventral sucker. The two intestinal limbs give off numerous branches, and extend to the posterior end of the body. The genital pore is situated between the two suckers, but nearer the ventral one. The eggs are 130-145  $\mu$

long, 70-90  $\mu$  broad, of a brown colour, and have an operculum at the anterior pole. These parasites inhabit the bile-ducts of herbivora and of man, producing large numbers of eggs which pass into the intestinal canal of the host. If an egg, after being expelled from the host, lies in water, the *Miracidium* develops, raises the operculum, escapes and swims about by means of its cilia until it meets its intermediate host, which is a small mollusc, *Limnæa truncatula*. It becomes lodged in the pulmonary cavity of this mollusc, and is then transformed into a *Sporocyst*. From germinative cells within the body cavity of the *Sporocyst*, *Rediæ* having a cylindrical form and a digestive canal are then developed. The *Rediæ* next wander out of the sac of the *Sporocyst* and migrate to some



other organ of the host. Each Redia further produces numerous *Cercariae*. The anterior portion of the body of a *Cercaria* resembles the adult trematode, whilst a long tail constitutes the posterior part of the body. The *Cercariae* eventually escape both from the Redia and from the intermediate host, and swim about until they become encysted on such objects as the leaves of aquatic plants or blades of grass. The definitive host is infected by ingestion of encysted *Cercariae* on such plants.



FIG. 7.—*Fasciola hepatica*, nat. size. (After Mosler and Peiper.)

The definitive host is usually the sheep or ox, less frequently the goat, horse, or other herbivora. In the former animals the parasite is common in Europe, Australia, and South America. The disease, "Liver rot," due to the presence of these flukes in the bile-ducts, is most common in marshy or ill-drained districts. The livers of affected sheep are reduced in size, markedly cirrhotic, the bile-ducts are much dilated, and contain both the parasites and their eggs in large numbers.

In man this trematode is a very rare parasite, for it is recorded in only twenty-three cases. As a rule, there have been in each patient only one or two examples of the parasite, and their presence has often resulted in little, if any, disturbance of the general health. In some of the cases, however, there were chronic symptoms, such as severe jaundice, hepatic enlargement, pain and tenderness, and hæmatemesis. In some instances the liver has contained a few of the parasites and been otherwise quite normal, in others there have been cirrhosis, dilatation of the bile-ducts, obstruction of the hepatic duct, and secondary conditions, such as gastritis and splenic enlargement.

*Fasciola hepatica*, var. *angusta* (Railliet, 1895).—One example, 25 mm. long, expectorated by a patient. Railliet regards it as a variety of *F. hepatica*, but Blanchard concludes that it is identical with *F. gigantea*, which was discovered by Cobbold (1854) in the liver of a giraffe.

*Dicrocoelium lanceolatum* (Rudolphi, 1803); Syn.: *Distomum lanceolatum* (Rudolphi, 1803).—The body is lancet-shaped, 8-10 mm. long and 1.5-2.5 mm. broad. The ventral sucker is situated behind the anterior at a distance equal to one-fifth of the length of the worm. The genital pore is situated in the median line between the suckers. The eggs are dark brown, 40-45  $\mu$  long, 22-30  $\mu$  broad, and have an operculum. The developmental cycle is imperfectly known. According to some the larval stage is passed in *Planorbis marginatus*. The adult trematode is a fairly common parasite in the bile-ducts of the sheep and ox, and is usually associated with *Fasciola hepatica*. It has also been found in some other animals, and there are seven recorded cases in which it has been found in the bile-ducts of man in Egypt, Germany, Italy, etc.

*Distomum oculi humani* (Aminon, 1833); Syn.: *D. ophthalmobium* (Diesing, 1850).—Four examples were found beneath the capsule of the lens in a child in Dresden, and are regarded as immature forms of some other species.

*Opisthorchis felineus* (Rivolta, 1885); Syn.: *Distomum felineum* (Rivolta, 1885); *D. conus* (Gurlt, 1831); *D. sibiricum* (Winogradoff, 1892).—It is 8-18 mm. long, 1.5-2.5 mm. broad, flattened, of a pinkish colour, and very transparent. The two cæca or limbs of the intestine are often filled with brown contents; the genital pore is just in front of the ventral sucker, the eggs



are oval, 26-30  $\mu$  in their longest diameter, and have an operculum. Little is yet known in regard to the development of this species, but the definitive host is supposed to be infected by ingestion of infected fish. Although the parasite has been found in various animals, it is most frequent in the cat, and inhabits the bile-ducts, causing their dilatation and more or less general cirrhosis of the liver. It is also parasitic in man; in Tomsk it has been found in nine cases, Winogradoff (1892) finding it eight times in a total of 124 autopsies. In none of the cases had the parasite caused the death of the patient, but in each instance had caused dilatation of the bile-ducts, thickening of their walls, cirrhosis and atrophy of the liver tissue, and in some cases there was jaundice and ascites. In many parts of the world this trematode is common in the cat. In Königsberg Braun found that 80 per cent were infected, and he therefore suggested that further instances of its occurrence in man would probably be recorded. The only case observed in Europe since then is that recorded by Askanazy (1900) in East Prussia, whilst in one other case the same writer found the eggs of this parasite. There is thus a total of eleven cases, the patients in each instance having been of the male sex.

*Opisthorchis conjunctus* (Cobbold, 1859); Syn.: *Distomum conjunctum* (Cobbold, 1859), is a small lancet-shaped trematode, about 10 mm. long and 2.5 mm. broad; its whole surface is covered with small spines. The genital pore is just anterior to the ventral sucker. The eggs are 34  $\mu$  long, 20  $\mu$  broad, and have an operculum. The developmental cycle is yet unknown. This species was obtained by Cobbold from an American fox, by T. R. Lewis (1872) from pariah dogs in India, and by M'Connell (1875-78) from two Mohammedans in Calcutta. It was found in the dilated and inflamed bile-ducts.

*Opisthorchis sinensis* (Cobbold, 1875); Syn.: *Distomum sinense* (Cobbold, 1875); *Distomum spathulatum* (Leuckart, 1876); etc.—It is lanceolate, 10-18 mm. long, 2-3 mm. broad, of a reddish colour and very transparent. The ventral sucker is situated behind the oral sucker, at a distance equal to one-quarter of the length of the body. The genital pore is just in front of the ventral sucker. The dark brown eggs measure 25-30  $\mu$  long, 15-17  $\mu$  broad, and have an operculum. The intermediate host is not yet known. This parasite was found by M'Connell in Calcutta in 1874, large numbers being seen blocking the bile-ducts of a Chinese patient. It has also been found in Mauritius, Corea, and more especially in Tonquin and Japan. Bælz (1883) first drew attention to its frequency in Japan, in some districts of which the disease due to the parasite is endemic, being characterised by dilatation of the bile-ducts and hepatic cirrhosis, and not infrequently causing the death of the patient.

*Opisthorchis Buski* (Lankester, 1857); Syn.: *Distomum Buski* (Lankester, 1857); *Distomum crassum* (Busk, 1859).—It is oval, 4-8 cm. long and 1.4-2 cm. broad. The ventral sucker is about 3 mm. behind the oral sucker, and the genital pore is between them. The eggs are 125  $\mu$  long and 75  $\mu$  broad, with an operculum. The mode of development is unascertained. The parasite has been found in about half-a-dozen cases in the fæces, small intestine, gall-bladder, and bile-ducts of native Chinese or of those who have dwelt in China.

*Opisthorchis Rathouisi* (Poirier, 1887); Syn.: *Distomum Rathouisi* (Poirier, 1887).—This species, which is regarded as probably identical with that last mentioned, was first found by Rathouis in the fæces of a Chinese subject, and was recorded by Poirier in 1887.



*Mesogonimus heterophyes* (v. Siebold, 1852); Syn.: *Distomum heterophyes* (v. Siebold, 1852) is oval, 1-1.5 mm. in length, 0.7 mm. in breadth, and reddish in colour. The genital orifices open at the bottom of a large genital sinus, which is situated behind and to the left of the ventral sucker. The eggs are reddish brown, 26  $\mu$  long and 15  $\mu$  broad. The mode of development is not known. The parasite has been found in a very few cases inhabiting the small intestine of persons in Egypt (Bilharz, 1851; Blanchard, 1891; Looss, 1894).

*Mesogonimus Westermanni* (Kerbert, 1878); Syn.: *Distomum Westermanni* (Kerbert, 1878); *D. Ringeri* (Cobbold, 1880); *D. pulmonale* (Bälz, 1883).—It is egg-shaped, 8-10 mm. long, 4-6 mm. broad, and of a reddish brown colour. The ventral sucker is about midway between the anterior and posterior extremities of the body, and behind it is the genital pore. The yellowish eggs are 80-100  $\mu$  long and 50  $\mu$  broad. Little is known of the development of this parasite, which is found in Japan and other eastern parts of Asia, where it is somewhat common. It is found in cavities within the lungs. These cavities may be as large as a walnut or a hen's egg, contain blood-tinged mucus, abundant eggs, and sometimes one or more adult worms, and usually communicate with a bronchus. The eggs have also been found in subperitoneal nodules and within inflammatory nodules in the brain.

The same species is parasitic in the cat, dog, and other animals in different parts of the world.

*Amphistomidae*.—The oral sucker is small, whilst the ventral sucker is large, and situated at or near to the posterior end of the body.

*Amphistomum hominis* (Lewis and M'Connell, 1876).—It is 5-8 mm. in length, 3-4 mm. in its greatest transverse diameter, and has a relatively very large ventral sucker, the transverse diameter of which is equal to half the total length of the worm. The eggs were 150  $\mu$  long, 72  $\mu$  broad, and had an operculum. This species, which has been observed on only two occasions, was found in large numbers adhering to the mucous membrane of the cæcum and colon of natives of India.

Other species of this family are parasitic in the stomach and intestine of the lower animals. *Gastrodiscus Sonsinoi*, for instance, is found in horses and mules in Egypt, Senegal, and Guadeloupe; *Amphistomum Hawkesii* is a common and sometimes fatal parasite of Indian elephants; others are found in the ox, sheep, and goat.

*Schistosomidae* are trematodes in which the sexes are separate.

*Schistosomum hæmatobium* (Bilharz, 1852); Syn.: *Distomum hæmatobium* (Bilharz, 1852); *Bilharzia hæmatobia* (Cobbold, 1860); etc.—In general form this worm resembles a nematode. The male is 5-16 mm. long, of a whitish colour, the oral sucker is apical, and close behind it is the posterior or ventral sucker. Behind this latter sucker the body is

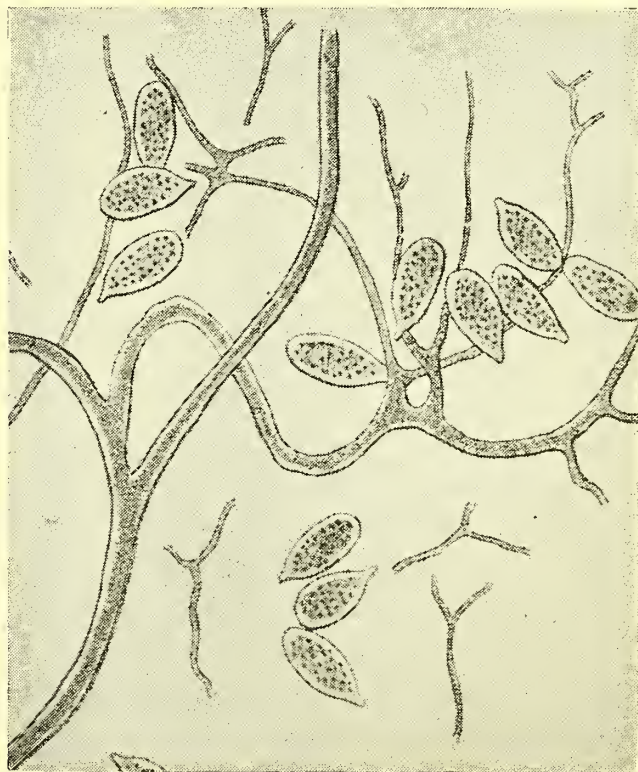


FIG. 8.—Surface view of vesical mucous membrane. Capillaries filled with blood corpuscles. Eggs of *Sch. hæmatobium* outside the capillaries. (After Lortet and Vialleton.)



expanded laterally, and the two marginal expansions thus formed are folded towards the ventral surface, and overlapping each other form the "gynæcophoric canal" in which the female is frequently contained. The genital orifice is at the anterior end of this canal just behind the ventral sucker. The female is almost cylindrical, 15-20 mm. long, thinner than the male, and with two suckers similarly situated to those of the male. The uterus opens just posterior to the ventral sucker. The eggs (Fig. 8) are oval, 130-200  $\mu$  long and 50-60  $\mu$  or more broad, each having a spinous projection at one pole, rarely at the lateral margin, but having no operculum.

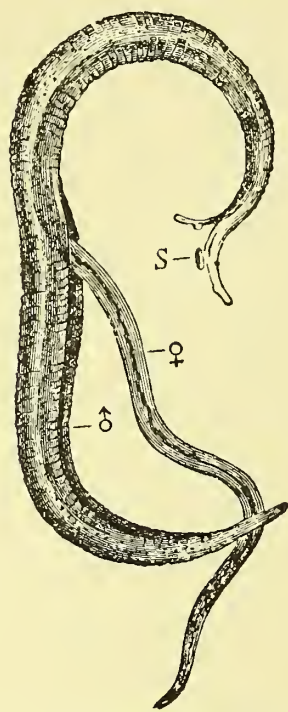


FIG. 9. — *Schistosomum hæmatobium*: S, posterior ventral sucker of male. (After Blanchard.)

The worm inhabits the smaller tributaries of the portal vein, is parasitic only in man and monkeys, and is mainly found in Africa and in the male sex. The disease it excites is endemic in Egypt, and especially so among the fellahs. According to Leuckart, Griesinger found the parasite 117 times in 363 autopsies, and Sonsino 13 times in 31 autopsies. Kaufmann (quoted by Lortet and Vialleton) has observed it in 40-50 per cent of autopsies on men, and in 13 per cent of those on women. Ruffer (1901) finds it in quite 50 per cent of all necropsies on natives in Egypt. In the eastern district of Cape

Colony, Natal, and the Transvaal Colony, the parasite is exceedingly common. Guillemard (1897), for instance, states that in Pietermaritzburg the majority of the male youths were apparently infected, and that in the Transvaal a large proportion of the male population suffer from the parasite. It is also found, though less frequently, along the whole of the east coast of Africa, on the borders of Lake Nyassa and of the Zambesi, in Tunis, the Gold Coast, Madagascar, Mauritius, and Arabia; and, as might be expected, cases are at times imported into other countries.

When the eggs are passed with the urine or feces, they each contain a ciliated embryo or *Miracidium*. If the eggs are allowed to remain in the urine or get into water containing decomposing animal matter the ovid embryo within each soon dies, but if the eggs are carried into pure water the embryos are soon hatched out, and are then of somewhat elongated form and actively motile. The subsequent stages of its life history are still uncertain, but as ingestion of free embryos by monkeys and other animals has not resulted in their being infected, it is probable that the life cycle is similar to that of *F. hepatica*, and that an encysted parasite is essential for infection of man. The actual mode of infection is likewise unknown, and although the frequency of the disease in Africa in those who are in the habit of bathing in fresh water points to the possibility of infection being brought about by penetration of the parasites along the urethra or rectum, it is more probable that persons are infected by ingestion of the parasites.

Further anatomical details of the worms and a number of experimental inoculations are recorded in the monograph of Lortet and Vialleton, *Etude sur le Bilh. hæmat. et la Bilharziose*, 1894.

The adult parasites live in large numbers within the tributaries of the portal vein. The females, which are in excess of the males, produce an abundance of eggs which block the portal capillaries and thus cause venous congestion and hæmorrhage. There is also a more or less diffuse infiltration of



eggs throughout the mucous and submucous coats of the bladder, rectum, or other pelvic viscus, with consequent inflammatory infiltration also. The changes are most pronounced in the bladder. The mucous membrane is covered with blood-tinged mucus, presents patches of hyperæmia and ecchymoses, and there is thickening partly of an inflammatory nature of the bladder wall. In cases of old-standing, papilliferous outgrowths or projections from the vesical mucous membrane are seen, and these projections, as well as the mucous membrane generally, are not infrequently infiltrated with urinary salts, chiefly urates and oxalates. Ulceration of the mucous membrane is not seen in all cases. Microscopically the eggs are found chiefly in the more superficial strata of the bladder wall, and around them is more or less extensive leucocytic infiltration. The earliest symptoms associated with these vesical lesions are pain and slight intermittent hæmaturia, which in course of time becomes more frequent and more constant. The frequency of micturition is increased, and the urine contains red blood corpuscles, pus cells, blood-clots, urinary crystals of various kinds, with much mucus, large numbers of eggs, epithelial cells, and débris. The disease is rarely confined to the bladder. The seminal vesicles and rectum are often affected, the ureters and kidneys less frequently so. In each instance the pathological conditions are essentially the same as those seen in the bladder. Involvement of the seminal vesicles is indicated by perineal pain and tenderness; infection of the rectum is either primary or secondary to infection of the bladder, and is characterised by symptoms similar to those of dysentery and by the presence of the eggs in the fæces; the spread of the disease to the ureters leads to pyelitis, hydronephrosis, pyelonephritis, and the formation of renal calculi. The eggs are seldom seen in the prostate, mesenteric glands, lungs, or liver, and have never been detected in the spleen, pancreas, or stomach.

The prognosis depends largely on the degree of infection; the most severe forms of the disease are observed in Egypt, and the patients often become markedly anæmic and wasted, and succumb to the disease. In many cases, however, in which there is not repeated reinfection, the inflammatory processes gradually subside and the patients regain their normal health. The only curative measures of avail are the removal, if possible, of the patient from the district in which the disease prevails, and such treatment as is directed to the relief of symptoms and the maintenance of the patient's strength.

*Schistosomum bovis* (*Sch. crassum*), which is parasitic in the portal tributaries of cattle and sheep in Egypt, Italy, Sicily, and possibly in India, is a different species from *Sch. hæmatobium* of man.

## CESTODES

*Tapeworms*.—These are flat worms without mouth or alimentary canal. Each tapeworm of man consists of a head or scolex, a neck, and a chain of proglottides or segments which, whilst remaining attached to one another, constitute a "strobilus." The whole tapeworm is therefore often regarded, not as a single animal, but as a polymorphic colony. The outer surface of a cestode consists of a cuticular membrane, within which is a parenchyma differentiated into a peripheral layer and a central mass, and in the parenchyma there are calcareous corpuscles and fusiform muscle fibres. For the adhesion of the worm to the host the muscle fibres in the head are specially developed, so as to form suckers or suckorial grooves, and in some species to form also an apical rostellum which has one or more circlets of hooks on its outer



surface. The youngest proglottides—those nearest the head—present no reproductive organs, but each of the older proglottides has both male and female sexual organs, the orifices of which open usually in close contiguity to one another at the bottom of a genital sinus, the orifice of which is termed the genital pore. In each proglottis there are numerous testes, whilst of the female reproductive organs that most readily recognised is the uterus, which in many of the tapeworms consists of a median canal with several lateral branches.<sup>1</sup>

*Development of the Cestodes.*—All the adult cestodes inhabit the intestinal canal of the definitive host and produce numerous eggs. Within each egg there develops a globular six-hooked embryo or oncosphere which, in the case of the Bothriocephalidæ, is covered with cilia. The eggs are evacuated with the fæces of the host, but further development occurs only after the embryo within its shell gains entrance into a suitable intermediate host. When carried into the stomach of this host the shell is digested, and the embryo being liberated penetrates the stomach wall, and either settles down in the body cavity of the host, or, as is more frequently the case, is carried by the venous or lymphatic channels to other parts of the body. In either case the embryo having reached its ultimate destination becomes transformed into a *larva*. In the Bothriocephalidæ the embryo develops directly into a larva which closely resembles the scolex of the adult worm. In all the other tapeworms of man the embryo is first transformed into a vesicle (less often into a solid mass), and within this one or more scolices or heads are, as it were, budded off. Some weeks or months are, as a rule, required for the complete development of the larva. If one scolex is formed from the wall of such a vesicle the whole larva is termed a *Cysticercus*, if more than one scolex is formed directly within a vesicle the larva is known as a *Cœnurus*, whereas if the scolices be formed from special sacs within the

vesicle the larva is designated an *Echinococcus*. If the scolex develops not within a true vesicle, but in a solid larva, this larva is known as a *Cysticercoid*. Cysticercoids are found solely in cold-blooded animals, especially in insects and molluscs. The scolex of each larva is a representation in miniature of the head of the adult tapeworm, and is usually retracted within the vesicle (Fig. 18), but if the scolex be protruded the vesicle appears as an appendage or “caudal bladder.” The cystic larvæ are therefore known as bladder worms. The size of the vesicle varies greatly in different species. *Cysticercus tenuicollis* of ruminants (the cystic larva of *Tænia marginata* of the dog) is often as large as a hen’s egg or an orange, *Cœnurus cerebralis* of sheep (the larva of *Tænia cœnurus* of the dog) may be as large as a hen’s egg, but *Cysticercus cellulosæ* of man is seldom an inch in its greatest diameter. If a larva be ingested by a suitable host the caudal vesicle is digested, the scolex is set free, and after attaching itself by its suckers and hooks to the intestinal



FIG. 10.—Head of *T. saginata* in contracted condition (× 8). (After Leuckart.)

mucous membrane it buds off successive proglottides from its distal extremity. Tapeworms are therefore acquired solely by ingestion of the associated larvæ.

Cestodes both in the adult and larval stages are harboured by man. There are a number of different adult tapeworms for consideration,

<sup>1</sup> The form of the uterus is readily seen if one or more proglottides are placed for a few minutes in a watch-glass containing glycerine, and then subjected to slight pressure between a slide and large cover-glass.



but only two larval species—*Cysticercus cellulosæ* and *Echinococcus polymorphus*.

*Tænia saginata* (Göze, 1782); Syn.: *T. mediocanellata* (Küchenmeister, 1855); *T. inermis* (Brera, 1802); etc.—This is the largest tænia found in man, and may attain a length of twelve to twenty-four feet or more. The head is somewhat cubical, measures 1.5-2 mm. in transverse diameter, has neither rostellum nor hooks, but has four hemispherical suckers, each of which is surrounded by blackish pigment. The thickness of the neck is less than that of the head. There are about 1000-1300 proglottides, larger than those of *T. solium*. Sexual maturity is attained at about the 500th segment.

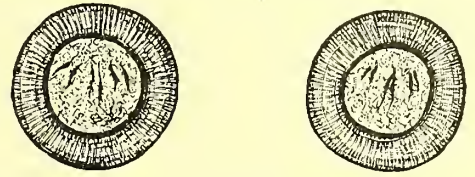


FIG. 11.—Eggs of *T. saginata*. (After Mosler and Peiper.)

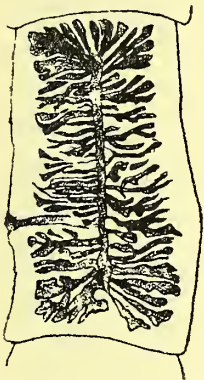


FIG. 12.—Ripe segment of *T. saginata* ( $\times 2$ ). After Leuckart.)

The mature separated segments are 16-20 mm. long, 4-8 mm. broad. The uterus has on each side about 20-35 lateral branches, each of which again branches dichotomously. The prominent genital papilla is situated laterally just behind the centre of each proglottis, alternating from right to left in successive proglottides, but often very irregularly arranged. The eggs, which closely resemble those of *T. solium*, are almost spherical, about 25-30  $\mu$  in diameter; the shell is thick, of a brownish yellow colour, has radiate markings, and each egg contains a six-hooked embryo or oncosphere. Malformations are not uncommon in this species; the most frequent is the prismatic or triangular strobilus, a transverse section of which resembles the letter Y.

Man is the sole host of the adult worm, which inhabits the intestinal canal. The head is firmly attached to the mucous membrane of the upper part of the small intestine, whilst the chain of proglottides lies in the lumen of the canal, the riper segments being nearest to the rectum. Persons as a rule do not harbour more than one example of this tapeworm, which may be found in persons of all ages. In all parts of the world it is a common parasite, and is certainly the most frequent tapeworm in Great Britain as well as in the whole of Western Europe. Its increasing frequency is often attributed to the fact that raw beef juice constitutes at present so frequent an article of diet in different diseases both of adults and children. The worm is exceedingly common in Eastern Europe, in Abyssinia, where almost every native is the host of this parasite, and in some of the States of South and Central America. The frequency is very largely dependent on the cleanliness and habits of the people.

The tapeworm is developed from *Cysticercus tæniæ saginatae*—*Cysticercus bovis*—which is parasitic almost solely in the muscles of the ox, very exceptionally in those of one or two other animal species, but practically never in those of man. It is solely by ingestion of the infected muscles of the ox, mealy beef, that man acquires the tapeworm, and it has been shown that it is not until about sixty days after ingestion of the cysticercus that mature proglottides commence to be discharged from the intestine. About six to ten proglottides are shed, singly or attached to one another, each day, and they very frequently migrate spontaneously through the anus. Each contains hundreds of eggs, and it is by water containing these that oxen are infected. After the shell has been digested in the stomach the embryo escapes, passes to the muscles, and in three to six months develops into a fully-formed cysticercus with one unarmed scolex which represents in miniature the head of the adult worm. The fully-developed *Cysticercus*



*bovis* is a small ovid bladder not more than 10-12 mm. long, and often no larger than a pin's head. Each is contained within a thin adventitious connective tissue capsule formed from the tissues of the host. On incising the cysticercus the fluid contained within escapes, and one can recognise the small and usually invaginated scolex attached to the middle of the long equator of the cyst. The small size of these cysticerci often renders their detection a matter of difficulty. Their favourite seats are the muscles of mastication and the heart, but they may be found in any muscle. They

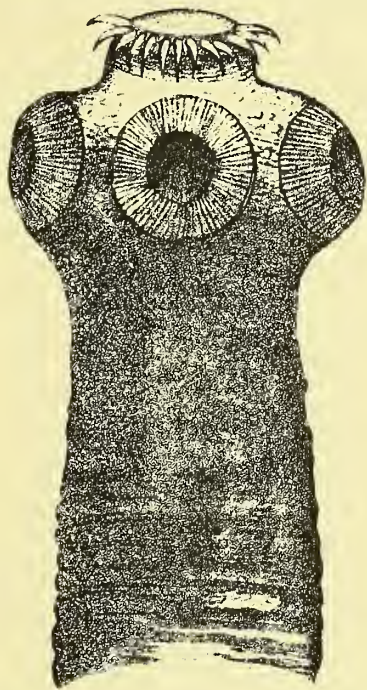


FIG. 13.—Head of *T. solium* ( $\times 45$ ).  
(After Leuckart.)



FIG. 14.—Eggs of *T. solium*. (After Mosler and Peiper.)

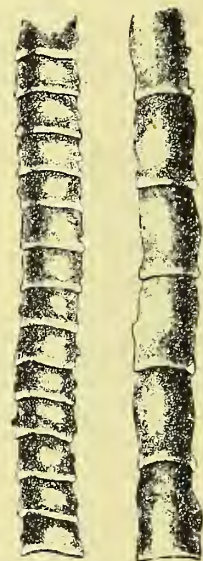


FIG. 15.—Half ripe and ripe segments of *T. solium* (nat. size).  
(After Leuckart.)

are exceedingly rare in any of the viscera but the heart. Some of the cysticerci often die spontaneously, and are then seen as small, hard calcareous nodules. The cystic parasites are killed at a temperature of  $47^{\circ}$ - $48^{\circ}$  C., but the temperature at the central portions of underdone roasts or steaks has never reached this point, and hence to eat beef only when it is thoroughly cooked is the surest means of prophylaxis.



FIG. 16.—Two proglottides of *T. solium* with uterus ( $\times 2$ ). (After Leuckart.)

*Taenia solium* (Rudolphi, 1810).—This tapeworm seldom exceeds 10 or 12 feet in length. The head is in general spherical, usually somewhat less than 1 mm. in diameter, and has anteriorly a rostellum with two circlets of hooks. The hooks number about 26 to 28 in all, and those of the inner, or anterior, circlet are the larger (Fig. 17). The head possesses four muscular suckers. The neck is thinner than the head, and gradually passes into the youngest of the proglottides. The total number of segments is about eight or nine hundred, though sexual maturity is attained about the 450th. The ripe segments at the end of the chain, which are seldom liberated singly, are 10 to 12 mm. in length, 5 to 6 mm. broad. On each side of the uterus are seven to eight lateral offshoots which do not branch so much as in the case of *T. saginata*. The genital papillæ alternate from right to left with greater regularity than in *T. saginata*, and the eggs resemble those of that tapeworm.

*Taenia solium* is found only in man. Its head is tenaciously fixed to the mucous membrane of the small intestine, often of the duodenum, and there



is seldom more than one example of the worm in any host. Persons of all ages may be the hosts of the tapeworm, which occurs in every country in the world. Although a common parasite, it is nowhere so frequent as *T. saginata*, and all records show that of late years it is becoming less and less frequent, in consequence, doubtless, of more stringent meat inspection in abattoirs.

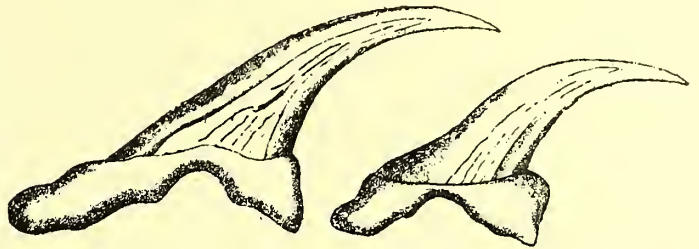


FIG. 17.—Larger (anterior) and smaller (posterior) hooks of *T. solium* ( $\times 280$ ). (After Leuckart.)

The cystic larva, *Cysticercus cellulosæ*, is found chiefly in the

pig—more rarely in other animals, for example, the dog and cat—or in man, inhabiting the intermuscular connective tissue, the brain, eyeball, or other structures. Man acquires the tapeworm solely by ingestion of measly pork,

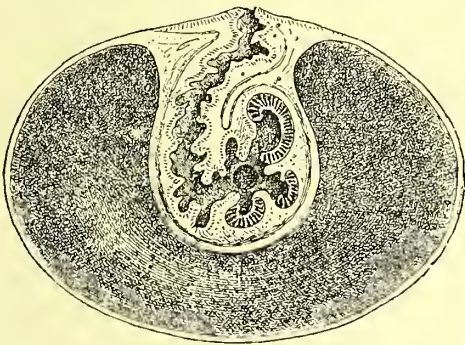


FIG. 18.—Median section through a cysticercus with fully formed scolex. (Braun after Leuckart.)

as the infected flesh of the pig is termed. There are no reliable statistics showing the frequency of measly pork in this country; it is probably, however, not very uncommon, and especially so in Ireland. The figures recorded by Braun show that in Prussia between 1878 and 1885 the number of infected pigs varied from 1 in 270 to 1 in 409. The larva, which is developed from an embryo of the tapeworm, is fully formed 10 to 12 weeks after ingestion of the egg. In the pig the cysticercus is a small elliptical bladder, 6 to 20 mm. in length, and 5 to 10 mm. in breadth. The scolex is situated at the middle of the long equator. The vitality of the larva is usually well maintained for some months at least, but cysticerci are not infrequently discovered dead and calcified. They are most numerous in the muscles, especially in the tongue and muscles of neck and shoulders, but are not frequent in the fat or in any of the internal organs except the heart. The cysticerci are, as a rule, killed at a temperature of  $47^{\circ}$  to  $49^{\circ}$  C., but whereas on thorough boiling the temperature at the central portions of the meat will exceed this by  $10^{\circ}$  to  $15^{\circ}$  C., and the cysticerci consequently be killed, the temperature at the similar portion of a moderately well-roasted joint has been found not to exceed  $46^{\circ}$  to  $48^{\circ}$  C., and the roast would therefore still be dangerous.

*Cysticercus cellulosæ* in the Human Subject.—Man acquires the cysticercus in consequence of the eggs of *T. solium* gaining entrance into the stomach. The infection may be brought about in more than one way. The eggs may be ingested with uncooked and imperfectly washed vegetables or fruit, or with drinking water polluted with sewage, or the eggs may be transmitted by the fingers from the anal region to the mouth, and, lastly, the proglottides or eggs may pass directly from the intestine into the stomach. The last two modes of infection are probably somewhat unusual, as in the great majority of persons who harbour the cystic larvæ there is no evidence of the presence of the tapeworm. The cysticercus is seen as a small, pale, yellowish, thin-walled vesicle, as a rule no larger than a pea, enclosed in a connective tissue capsule, and containing clear, almost colourless watery fluid. The scolex appears projecting within the cavity, and is about the size of a millet seed, and, if thus projecting into the cavity of the vesicle, has its four suckers, rostellum, and hooks in-



vaginated, and they cannot therefore be seen until pressure is exerted, so as to cause extrusion of the head. At times the cysticercus is as large as a cherry or a grape, but only when it has been subjected to but little pressure during its growth—for example, if growing into the ventricles of the brain. It is not often that the cysticercus, losing its globular form, becomes irregular or racemose, or that it is capable of spontaneous movement. The cysticerci may live in the human tissues for ten or twenty years. The frequency of the parasite is not great in any country, and in the United Kingdom it is not often observed. The majority of cases are recorded in the male sex, and during the third and fourth decade. The number of cysticerci present varies enormously in different cases—there may be only one or two, or perhaps thousands. The most frequent sites are the brain and the eyeball, less frequent are the voluntary muscles, the heart, subcutaneous connective tissue, other internal viscera, etc.

*In the brain* the cysticerci, as already mentioned, may attain a considerable size, and the connective tissue capsule is thin. They are most frequent in the pia and arachnoid mater and in the vicinity of the fissure of Sylvius, or may be situated in the ventricles, but are infrequent within the substance of the brain. The cysticercus has usually been solitary, and the surrounding brain substance has often been healthy, though in other instances it has manifested signs of chronic inflammation. The resulting symptoms depend largely on the number and anatomical sites of the cysticerci. Not infrequently there have been no symptoms whatever during life; in other cases there have been headache, vertigo, diplopia, optic neuritis, hydrocephalus, and other cerebral phenomena.

Intra-ocular cysticerci have been most frequent in Germany. The parasite is usually subretinal, may cause detachment of the retina, and may penetrate through into the vitreous. Subconjunctival cysticerci are still less common, and in the areolar tissue of the orbit they are exceedingly rare.

The heart is not often infected, although in rare instances the myocardium has been extensively infected, but the condition gives rise to no symptoms which enable a diagnosis to be made during life. General infection of the muscles and subcutaneous tissue is exceedingly rare; it causes symptoms resembling those of myositis or extensive peripheral neuritis.

An increase in the number of eosinophile leucocytes may be expected. Achard and Loeper (1900), recording this condition in one case, found that injection of the fluid contained within the cysticercus into a mouse increased the number of its eosinophile leucocytes.

A clinical diagnosis can only be made when the cysticercus is within the eye, or is situated subcutaneously, and can be excised; the prognosis depends on the site and number of the larvæ, and surgical treatment alone is of value.

*Tænia africana* (von Linstow, 1900); two imperfect examples found in German East Africa. The head is unarmed, the neck is thick, and the genital pores alternate irregularly from side to side.

*Tænia asiatica* (von Linstow, 1901); one example found in Asiatic Russia, 298 mm. long. The genital pores are all on one lateral margin.

*Dipylidium caninum* (Linné, 1758); Syn.: *Tænia canina* (Linné, 1758); *Tænia cucumerina* (Bloch, 1782); etc.—This worm is 10 to 40 cm. in length, the head has a rostellum, four circlets of small hooks, and four suckers. The ripe proglottides have the shape of melon seeds, and each has a double reproductive system with two genital pores, one on either lateral margin. It is a common parasite of the dog and cat, and has occasionally been found in children. There have usually been several examples of the worm in the



individual cases. The cysticeroid larva, *Cryptocystis trichodectis*, is found in the common dog flea, seldom in the dog louse, or in *Pulex irritans* of man.

*Hymenolepsis nana* (v. Siebold, 1852); Syn.: *Tænia nana* (von Siebold, 1852); etc.—It is about 1-2 cm. in length, and 0.7 cm. in its greatest transverse diameter. The head has a rostellum, one circlet of 24-30 small hooks, and four suckers. There are about 200 proglottides in all. The genital pores are all situated on the same lateral aspect of the chain. The worms are usually multiple, and penetrate into the intestinal mucous membrane. They are rare, except in Italy and Sicily, where they are not at all uncommon. This parasite is regarded by some as identical with *Hymenolepsis murina*—a tapeworm of the rat and mouse, and the larva of which is a cysticeroid found in the same host as the adult worm.

*Hymenolepsis diminuta* (Rudolphi, 1819); Syn.: *Tænia diminuta* (Rudolphi, 1819); *T. flavopunctata* (Weinland, 1858); etc.—It is 20-60 cm. long, 3.5 mm. broad, the head is unarmed, and the genital pores are all on the same lateral aspect of the chain. It is a common parasite in the rat, mouse, and other rodents, and has been detected in one or two instances in children. The cysticeroid larva is found in certain insects: in beetles, in *Asopia farinalis* both in the butterfly and caterpillar stages, and in *Anisoblabis annulipes*—one of the Orthoptera.

*Davainea madagascariensis* (Davaine, 1869); Syn.: *Tænia madagascariensis* (Davaine, 1869).—This cestode is 25-30 cm. in length, and has 500-700 proglottides. The head has two circlets of hooks. The genital pores are all on the one lateral margin of the worm. It has been found in a few cases in Madagascar, Mauritius, and Bangkok. The larval stage is not known.

*Bothriocephalus latus* (Bremser, 1819); Syn.: *Dibothriocephalus vulgaris* (Linné, 1758).—This parasite has a length of 16-26 feet, and is consequently the largest human tapeworm. The head is oval or almond-shaped, 2-3 mm. long, has neither suckers nor hooks, but has a deep furrow or groove on each side, corresponding to the dorsal and ventral aspects of the proglottides. The neck is of variable length, and behind it there are 3000-4000 proglottides, sexual maturity being attained about the 600th. The breadth of the proglottides much exceeds their length, except towards the far end of the chain from the head, when they become longer and narrower. The genital pore of each proglottis is situated in the median line of the ventral surface, the

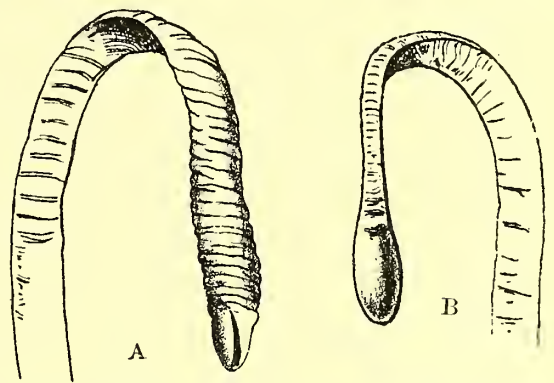


FIG. 19.—Head of *Bothriocephalus latus* (×8). A, from the flat side; B, from the margin. (After Leuckart.)



FIG. 20.—Eggs of *Bothriocephalus latus*. (After Mosler and Peiper.)

orifice of the genital cloaca being in front of the smaller uterine aperture. The uterus is a simple convoluted tube, the convolutions being arranged so as to resemble a rosette with from four to seven loops. The testes number about 700, and are situated in the lateral portions of the proglottis. The eggs are oval, of a brown colour, 70  $\mu$  long by 45  $\mu$  broad, and the shell has an operculum at one pole. They are shed from the proglottides before the latter are detached from the chain. If an egg has been passed from the host and carried into water, a six-hooked embryo gradually develops



within the shell in the course of several months. The embryo, which is covered with cilia, pushing open the operculum, then escapes and swims about. Its intermediate hosts are, as was proved by Braun, the pike and the burbot, though the larvæ may be seen in other fishes also. The larva, which is 1-2.5 cm. long, 2-3 mm. broad, and has a head exactly similar to that of *B. latus*, inhabits the muscles, viscera, and peritoneal cavity. When the larva is ingested by man the head buds off proglottides, and the adult tapeworm is thus formed.

*Bothriocephalus latus* has a definite geographical distribution. It is found in the western cantons of Switzerland and the adjacent parts of northern Italy. It occurs also in the districts bordering on the Baltic, especially the eastern provinces of Prussia, and the Swedish and Russian coasts, and again is a frequent parasite in Japan. In all these countries the tapeworm is acquired by eating raw, though often smoked fish. It is a tapeworm very seldom seen in this country.

*Bothriocephalus cristatus* (Davaine, 1874).—This worm is described as having no furrows on the head, and as presenting a difference from *B. latus* as regards the uterine "rosette." It is, however, not a separate species, but merely an anomalous form of the latter worm (Blanchard, 1896; Galli-Valerio, 1900; etc.).

*Bothriocephalus cordatus* (Leuckart, 1863).—It is 80-115 cm. long, the head is heart-shaped, and has two grooves, the genital pore lies in the median line of each proglottis. It is parasitic in Iceland and Greenland in the dog and walrus, and occasionally in man.

*Bothriocephalus Mansonii* (Cobbold, 1883); Syn.: *Ligula Mansonii* (Cobbold, 1883), etc.—Only the larva is known. It may be as much as 20 cm. long, and 2-8 mm. broad. The head has two grooves. It has been found in a few cases in China and Japan, inhabiting the connective tissues.

*Symptoms caused by Tapeworms.*—The head is firmly fixed to the mucous membrane between the villi, and no doubt is often buried to some extent in the mucous membrane. The parasite consequently causes some amount of irritation and catarrhal inflammation. In most cases, however, the disturbance caused is insufficient to produce any symptoms, the first indication of the worm being the passage of proglottides per rectum. In other cases the most common symptoms are desire for an excessive quantity of food, great partiality for sweetened articles, intermittent colic, and other unpleasant or painful abdominal sensations, especially towards the umbilical region; further, diarrhoea, anal pruritus, and in rare instances vomiting, and nervous phenomena similar to those seen in infection with *Ascarides*—for example, headache, inequality in the size of the pupils, convulsions, paresis, and so forth.<sup>1</sup> In some cases, again, there is considerable anæmia, but this is rarely serious except in infection with *Bothriocephalus latus*, when the anæmia has been recorded as of the pernicious form. None of the symptoms are in any way diagnostic; the only certain proof of the presence of a tapeworm is the detection of proglottides or ova. The former rarely migrate spontaneously unless they be those of *T. saginata*, in which species, too, they are larger and more often single than is the case with *T. solium*. In *B. latus* long chains of segments are passed. The especial points to be relied on in diagnosis of the species from one another are the characters of the uterus and the structural features of the head. If no proglottides are being

<sup>1</sup> These symptoms are probably due to toxic products formed by the cestodes. Calamida (*Centr. f. Bakt.* xxx., 1901) shows that the toxins obtained from *Dipyl. caninum* and *T. cœnurus*, when injected into rabbits and guinea-pigs, are not only pathogenetic, but that they cause hæmolysis and are positively chemiotatic, especially as regards the eosinophile leucocytes.



expelled by the patient, the characteristic eggs should be sought for; but those of *T. saginata* can with difficulty be distinguished from those of *T. solium*.

*Treatment.*—The day before the administration of the anthelmintic, the patient receives a light and liquid diet, and takes at night a dose of castor oil or other cathartic. The anthelmintic is then given on the following morning to the fasting patient. Filix mas is the best drug; for an adult 60-120 minims of the liquid extract are given in combination with mucilage and syrup in peppermint water, etc., or it is given in capsules. Two hours later castor oil is taken in order that the worm may be expelled. It is most important to search for the scolex, for unless it be expelled the development of proglottides will continue as before. If only part of the tapeworm have been expelled, and part be still protruding from the anus, no attempt should be made to pull the worm out, for a simple rectal injection will cause its expulsion with greater certainty, and is followed by less risk of tearing it and of leaving the scolex within the rectum. Cusso, kamala, pelletierin, turpentine or naphthalin, may be used instead of filix mas, but are not more efficacious. After the expulsion of a tapeworm, the possibility of there being more than one example of the parasite, and also of there being examples of different species simultaneously in the intestine, should not be overlooked; and, lastly, as an important means of prophylaxis, the expelled worm should be destroyed by burning.

*Echinococcus disease*—the condition resulting from the presence, in the tissues, of *Echinococcus polymorphus*, the larva of *T. echinococcus*, an intestinal cestode of the dog—has been already dealt with under the headings of “Hydatid disease” and “Liver.” Only two or three points in the recent literature will therefore be referred to. As to the nature of the bacteria which induce suppuration within the cyst little is yet known, but the causal bacteria are probably in many instances anaerobes. Hallé and Bacaloglu (1900), for example, examining a foetid hepatic echinococcus cyst, found two anaerobes (*Staphylococcus parvulus* and *Bacillus fragilis*) both very abundantly, and two aerobes (*Streptococcus pyogenes* and *Bacterium coli*) both very scantily. Our knowledge of multilocular echinococci has been much advanced by the monograph of Melnikow-Raswedenkow, “*Studien u. d. Echinoc. alveolaris sive multilocularis*” (1901). The primary convoluted chitinous cyst, says this writer, has granular protoplasm both on its inner and outer surface, from both of which there arise three kinds of embryos, finely granular “young forms,” encapsuled ovoid embryos, and scolices. He indicates the close analogy between the development, within the human tissues, of the alveolar (multilocular) echinococcus and that of certain trematodes; and, lastly, he refers in detail to the metastases in the lungs, brain, and elsewhere. Melnikow-Raswedenkow brings the number of recorded cases of multilocular echinococcus up to 235, but since then at least two new cases have been reported (G. Hauser; and Rénon, 1901). In Hauser’s case there was a primary multilocular echinococcus of the right lung and pleura with multiple metastases more especially in the brain. The case is recorded in *Festschr. d. Univ. Erlangen f. Prinzreg. Luitpold*.

(B) NEMATHELMINTHS (*νήμα*, thread), the round worms. Of these there are two orders: Nematodes and Acanthocephali. The former have an alimentary canal, the latter have none.

#### NEMATODES

The nematodes or round worms are cylindrical and elongated, and in general are filiform or fusiform in shape. Each has a well-developed



alimentary tract, commencing in a mouth at the anterior extremity which is usually pointed, and terminating at the anus, which is usually situated some little distance from the posterior end of the worm. The sexes are almost invariably separate. The males are smaller than the females, and the posterior extremity of the former is often ventrally curved. The chief anatomical features are the following: The outer surface is formed by a cuticula which often shows fine transverse striæ, and beneath this are from without inwards, an epithelial layer, the hypodermis, and the cutis. The cutis is markedly thickened on each lateral aspect to form the "lateral lines," the less prominent thickenings of the cutis anteriorly and posteriorly constituting the "median lines." Within the cutis is the muscular layer, the cells of which usually have the form of large vesicles. The oral aperture is often surrounded by lips or papillæ, the mouth sometimes presents cuticular "teeth," and behind the mouth there are in succession œsophagus, stomach, middle intestine, and posterior intestine, the latter opening at the anus. The nematodes possess a well-developed nervous system, and there are numerous papillæ at the posterior extremity of the males. Both sexes have a system of excretory vessels, of which the canals in the "lateral lines" are easily recognised. The male has a single straight or convoluted testicle, the genital aperture opens into the posterior intestine, which is therefore a cloaca, and there are two spicules—sometimes only one—capable of being protruded from the cloacal aperture. The vulva of the female is situated ventrally, usually in the anterior half of the body, and connected by a short vagina with two uteri. The ovaries are usually long and much convoluted. The ova are fertilised within the uterus, are enclosed by a more or less thick shell, outside which there is sometimes an albuminous sheath. In most species the females lay eggs, each containing a fully developed or as yet imperfectly developed embryo; in a few species the embryo escapes from the egg before being discharged from the maternal body. The host is infected by an embryo, either in a free state or enclosed within its shell (direct infection); or the embryo first infects an intermediate host, and the definitive host is thus indirectly infected.

The parasitic nematodes which have to be considered belong to one or other of the following families: Angiostomidæ, Filariidæ, Trichotrachelidæ, Strongylidæ, and Ascaridæ.

ANGIOSTOMIDÆ.—*Strongyloides intestinalis* (Bavay, 1877); Syn.: *Rhabdonema strongyloides* (Leuckart, 1883); etc.—The worms of the parasitic generation, *Anguillulæ intestinalis*, are about 2·2 mm. long and 0·03 mm. broad. They live in the upper part of the small intestine. According to Askanazy (1900) they bore into the mucous membrane, there the females deposit their eggs, and the embryos, which are about half as large as the adults, escape and gain the lumen of the intestine, to be afterwards evacuated with the fæces of the host. The second generation, *Anguillula stercoralis*, has a free existence: in foul water the embryos discharged from the host become mature, the females are oviparous, whilst the subsequent embryos are again of the same form as the parasitic generation, and if ingested by a suitable host develop into the mature *Anguillulæ intestinalis*.

The parasite is found in Cochin China, Ceylon, Africa, Brazil, Italy, Spain, and other parts of Europe. Strong has recorded a case in the United States (*J. Hopk. Hosp. Rep.*, x. 1901), and gives many bibliographical references. The parasite is not of great pathogenicity, having been detected in apparently healthy persons, but in tropical and subtropical climates it is present in great numbers in some cases of enteritis, and is not infrequently associated with *Ankylostoma duodenale*. As the eggs of the



parasitic generation are uncommon in the fæces diagnosis will, as a rule, be possible only after detection of the embryos in the stools.

FILARIIDÆ.—The members of this family, and the morbid conditions they give rise to, have already been considered in vol. iii.

TRICHOTRACHELIDÆ.—*Trichocephalus trichiurus* (Linné, 1771); Syn.: *Trichocephalus hominis* (Schränk, 1788); *T. dispar* (Rudolphi, 1801); etc.; whip worm; is of a pinkish yellow colour. The male is 40-45 mm. long, the female about 50 mm. long; in each the anterior three-fifths of the body is long and thin like the lash of a whip, the posterior two-fifths being much thicker (about 1 mm.) and in the male spirally curved. The male has only one spicule. The eggs are oval, possess a brown shell, within which is a thick doubled contoured membrane prolonged at each pole to form a prominent and characteristic projection. The human subject is almost the only host. The parasite lives in the cæcum and adjacent parts of the large intestine, being present usually in scanty, but sometimes in large numbers. The thin anterior portion of the worm, containing the oesophagus, is usually buried in the mucosa. The parasite is found in all parts of the world, in persons of all ages, and is probably a very common parasite, more particularly in Italy and Egypt. The eggs are passed with the fæces, and the embryos then develop. Infection occurs by eggs containing embryos being conveyed to the mouth with the food, or by means of the hands when soiled with contaminated earth, etc.

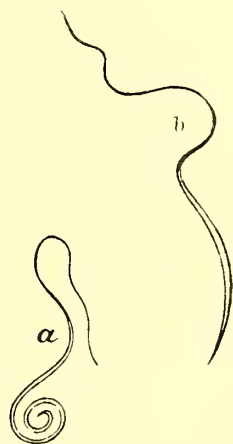


FIG. 21.—*Trichocephalus hominis*: a, male; b, female; nat. size. (Manson, after Blanchard.)

In many instances the trichocephalus causes no symptoms, being probably present in scanty numbers; in other cases, when probably many of the parasites are present, it causes profuse, long continued, and intractable diarrhœa, to which considerable anæmia and wasting are superadded. The cause of the anæmia is to some extent indicated by the presence of iron pigment in the intestinal epithelium of the worms. The stools are loose, yellowish, contain abundant characteristic eggs, and not unfrequently blood

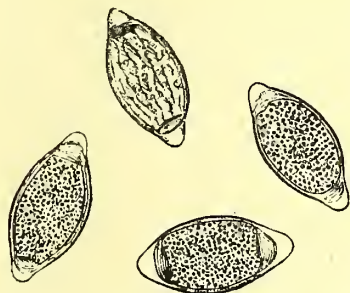


FIG. 22.—Eggs of *Trichocephalus hominis*. (After Mosler and Peiper.)

and Charcot-Leyden crystals, the latter, however, being of no diagnostic significance. Metschnikoff (1901) shows that the trichocephali can also cause symptoms resembling those of appendicitis, and J. Girard (1901) in one case found the parasites in the vermiform appendix, causing appendicitis. The diagnosis is easy if the stools be examined microscopically. In a case of mild infection the prognosis is good, but in a severe case is somewhat unfavourable. Treatment is best carried out by the use of thymol. In the morning when fasting thirty grains

are given in capsule, and a strong purgative in the evening. This is repeated on the two following days. Peiper recommends benzine internally as well as enemata containing the same drug.

Many species of trichocephalus are parasitic in the lower animals. *T. affinis*, for example, is found in the ox, goat, and sheep; *T. crenatus*, which Leuckart regarded as identical with *T. hominis*, is parasitic in the pig; and *T. unguiculatus* in the rabbit and hare. They are common parasites, but are not considered to be pathogenetic.

*Trichina spiralis* (Owen, 1835) is parasitic in two forms: (a) the sexually mature adults in the small intestine; (b) the immature embryos or larvæ



encapsuled in the voluntary muscles. *The adult trichinae*.—The male is 1.5 mm. long and 0.04 mm. broad, the anterior end is pointed, the posterior

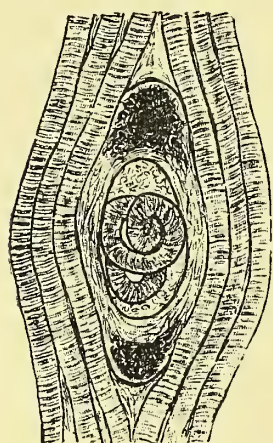


FIG. 23.—Trichinae encapsuled in muscle. (After Mosler and Peiper.)

extremity is broader, has no spicules, but has two conical appendages, one on either side of the cloaca. The female is 3-4 mm. long and 0.05-0.06 mm. broad, and viviparous. The vulva is situated in the anterior fifth of the body. The fertilised ova having passed into the uterus become mature, and the embryos are then hatched out and thereafter escape from the body of the adult female. The embryos at birth are 90-100  $\mu$  long and 6  $\mu$  broad. They bore through the intestinal wall, and thence reach the voluntary muscles. The embryos when seen as *muscle trichinae* have attained a length of 0.7-1 mm., are encapsuled, and usually spirally coiled.

The three most important hosts are the rat, pig, and man. The mouse, dog, cat, fox, and other animals are less frequently affected; the horse, ox, sheep, guinea-pig, etc., may be infected artificially. The muscles of any animal containing encapsuled trichinae are termed trichinous. Man acquires *Trichina spiralis* by the ingestion of trichinous pork, and similarly the pig, rat, or dog acquires the parasite by eating the trichinous flesh of other animals.

Trichiniasis, or trichinosis, is a disease which occurs in all countries. The two most important factors in causing the disease in man are: firstly, the prevalence of the disease in the lower animals, more particularly in the pig; and, secondly, and of greater importance, is the partiality for raw ham or sausage or for underdone pork manifested by persons of any nationality. In view of this latter factor the disease is, as might be expected, much more frequent in Germany, and more especially in Northern Germany, than elsewhere in Europe; and from almost whatever part of the world statistics are taken, it is found that a relatively large number of infected persons have been Germans.

Friedberger and Fröhner (1896) calculate that in Germany of 10,000 swine one is infected with trichinae; or again, that at the abattoirs in Prussia there were 10,000 affected swine during 1876-82, and 3000 during 1890-93. In some parts of Germany, too, infection is probably conveyed to man by dog's flesh. Pirl (1899), for example, records that in Dessau there are on an average over 200 dogs slaughtered for food every year, and that of 405 two were markedly infected.

The disease is seldom seen in this country, nor is it common in North America. Osler (1898), for instance, says that up to 1893 there had been only 709 cases in the United States, with, however, records of "40 or 50" further cases up to February 1898. Yet the disease in the pig is exceedingly common in the United States, though American writers seldom admit the fact. At Hamburg in 1883, 0.69 per cent of American pork was found to be trichinous, whilst Neumann (1892) quotes Chatten, who at Havre found 14.65 per cent infected, and he considers that on an average about 3 per cent of pork exported from America contains trichinae.

Trichinous flesh having been ingested, the capsule surrounding each embryo is digested in the stomach, and the embryo thus liberated. Two or four days later each embryo has developed in the intestinal canal of the host into an adult male or female trichina, and now inhabits the upper part of the small intestine. After fertilisation of the ova of the female a fresh



brood of embryos is produced, the liberation of these embryos from the adult female commencing seven to eight days after ingestion of the trichinous flesh. The birth of embryos, to the number of about 1000 to 1500, continues for about five or six weeks, after which time the adult trichinae usually die and are expelled from the host. The embryos, which are 90-100  $\mu$  long, penetrate the intestinal wall of the host and wander thence, probably into the lymphatic vessels, and are then carried by the thoracic duct to the right side of the heart, and being carried through the pulmonary capillaries are arrested in the capillaries of the voluntary muscles. According to Askanazy (1894) and Cerfontaine (1893), however, the female trichinae wander into the mucosa and submucosa, and then deposit their embryos in the lymphatic spaces. In the muscles the embryos attain the length of about 1 mm. Eight to ten days are required before this migration is accomplished, and infection of the muscles is therefore not complete until fifteen or sixteen days after the ingestion of trichinous flesh. The developmental cycle is thus completed. Ingestion of trichinous flesh is essential if an animal is to be infected with *Trichina spiralis*, for unless the embryos pass through an intermediate or larval stage in the muscles they do not attain maturity in the intestinal canal.

Any voluntary muscle may be affected, but the trichinae are always most numerous towards the junction of a muscle and its tendon. The muscles most abundantly infected are in order of frequency the diaphragm, intercostals, cervical, laryngeal and ocular muscles, biceps and triceps. The total number of trichinae present in the muscles in any given case is probably very great, 30-100 millions on an approximate calculation.

The embryo, when it has ceased its migration, becomes coiled up in a wide spiral within the sarcolemma. The presence of the parasites causes acute interstitial myositis, and a capsule is thereby formed around the trichinae. This capsule, formed of connective tissue, sarcolemma, and of a layer derived from the parasite, is oval or lemon-shaped, its long axis is parallel to that of the muscle fibres, and it measures about 0.4 mm. long, by 0.2 mm. in diameter. Within a capsule there is usually one embryo, sometimes two or more. The muscle fibres undergo granular degeneration and fragmentation, and show nuclear proliferation. Eosinophile leucocytes are found in relative abundance in the affected areas. There is also a great increase of eosinophile leucocytes in the peripheral blood, a point of very considerable diagnostic importance. T. R. Brown (1898) records a case where they formed 68.2 per cent of the total leucocytes. The parasite, when once encapsuled, does not exhibit any further change, and yet is capable of retaining its vitality for many years. The capsule, however, frequently undergoes secondary changes. Of these the most important is calcification, which occurs first at the poles of the capsule. This change is often deferred for years, more especially in the pig, and in man it rarely occurs before the fifth month, and in the pig until after the seventh month.

Even within a calcified capsule the trichina often preserves its vitality for years. Other, though less important, changes are hyaline degeneration and the formation of adipose tissue, which occurs towards the poles of the capsule.

*Trichinous Pork.*—If the pig die or be killed within six weeks of infection, one finds signs of acute enteritis and both adult and embryonic trichinae in the intestine. At a later stage the trichinae are found only in the flesh. To the naked eye small scattered greyish points are seen, but are not readily detected unless calcification has occurred. They are most likely to be confounded with *Sarcocystis Miescheri* (*vide* p. 204); the cyst of the latter,



however, is usually unmistakably larger than are the capsules of trichinae. For diagnosis it is advisable that small fragments of the flesh should be cut out, placed on a slide, teased out or subjected to pressure, 0·1 per cent acetic acid solution added, and the specimen examined with a magnification of about 40. Portions of the flesh of all pigs slaughtered in Germany are subjected to systematic examination of this nature. If larval trichinae are found the whole carcase should be condemned. It may be important, however, to ascertain whether the trichinae be alive or dead. To do so, the slide bearing the teased out muscle is heated to 35°-40° C., and if the trichinae are alive they will exhibit more or less active movement. A more reliable method is that of feeding a sparrow with the suspected flesh. In birds the larval trichinae become sexually mature, and produce a fresh brood of embryos, but the latter do not migrate to the muscles, but are expelled with the faeces. The sparrow is therefore killed about ten hours later, and its intestinal contents examined microscopically. Were the trichinae alive when ingested, they are now seen in active movement on the slide; but were they dead, they are now motionless and uncoiled, or perhaps only fragments of the dead worms have resisted digestion.

Larval trichinae soon perish if subjected to heat. Krabbe (quoted by Neumann), for example, found that small pieces of infected pork were rendered innocuous by immersion in water at 55° C. for five minutes, at 54° C. for ten to fifteen minutes, and at 52·5° C. for twenty-five to thirty minutes. In actual practice thirty-six minutes' boiling for each kilogram of weight is found to kill all trichinae with certainty. Again, as with cysticerci, roasting is less reliable than boiling, for unless the central portions of the joint be thoroughly cooked, the trichinae will retain their vitality. Ordinary freezing has no effect on trichinae, and salting and smoking, though often efficacious, are both unreliable. The prophylactic measures to be employed against trichiniasis are, therefore, the systematic examination of the flesh of all pigs slaughtered, and, what is still more important, because such examination is not entirely reliable, the thorough cooking of all pork, ham, or sausage.

The clinical features of trichiniasis in the human subject are dealt with in the article "Muscles," vol. viii.

STRONGYLIDÆ are all characterised by having six oral papillæ, and by the males having a caudal bursa. Representatives of three genera, *Eustrongylus*, *Strongylus*, and *Uncinaria*, have to be considered.

*Eustrongylus visceralis* (Gmelin, 1789); Syn.: *Eustrongylus gigas* (Rudolphi, 1802) is of a blood-red colour. The male measures as much as 40 cm. in length, 4-6 cm. in transverse diameter, and has one spicule. The female may be 100 cm. long and is oviparous. It is a parasite of the pelvis of the kidney which it destroys. It is chiefly seen in the dog, less frequently in the horse, ox, or other animal, and in a few isolated cases has been discovered in man.

*Strongylus subtilis* (Looss, 1895).—This species has been found in the small intestine of native inhabitants in Cairo and Alexandria. The male is 4-5 mm. long, and has two spicules; the female is 5-7 mm. long, and oviparous.

Strongyli are very frequent parasites in the bronchi of the lower animals. There are eight different species, and all are pathogenetic. The most important are *Str. filaria* in the sheep and goat, *Str. micrurus* in bovine animals, and *Str. paradoxus* in the pig. *Str. paradoxus* has in one or two instances been detected in man. Lastly, there is *Str. rufescens* which inhabits the bronchi of sheep and goats, the eggs and embryos infecting



the lungs and causing extensive broncho-pneumonia or nodular lesions resembling tuberculosis. The disease is very common and fatal in lambs during spring and early summer. There is no analogous condition in the human subject.

*Uncinaria duodenalis* (Dubini, 1843); Syn.: *Ankylostoma duodenale* (Dubini, 1843); *Sclerostoma duodenale* (Cobbold, 1864); *Dochmius duodenalis* (Leuckart, 1876); etc.—The mouth of each worm bears six chitinous teeth. The male is 6-10 mm. long, and at the posterior extremity has a trilobed, somewhat bell-shaped bursa with two long slender spicules. The female is on an average 11.4 mm. in length, the vulva is in the posterior third of the body, and the caudal extremity is pointed. The eggs are oval, about 50  $\mu$  long by 35  $\mu$  broad, and thin shelled. At a suitable temperature (20°-30° C.) and in the presence of decomposing faecal matter the actively motile embryos are hatched out. Although it is not yet definitely determined whether there is a free generation, there is considerable evidence against such a supposition, and Giles (1901) admits that his theory of an alternation of generations is untenable.

Man is probably the only host of this worm, although the same species is said to be parasitic in certain monkeys. The parasite inhabits the duodenum and upper parts of the small intestine, the anterior extremity being imbedded in the mucous membrane and fixed thereto by the chitinous teeth. It is a frequent parasite in tropical and subtropical climates, is exceedingly common in Egypt, Burmah, Madras, Bengal, and other parts of India, in the West Indies, Brazil, San Salvador, and Guatemala. It is common, too, in Italy, and by Italian labourers has been transferred to other parts of Europe. Severe outbreaks of ankylostomiasis occurred during the tunnelling of the St. Gothard Pass, and still occur from time to time in mines, brickworks, etc. In northern Europe, however, the disease is not endemic, for the free living embryos are killed by frost. Perhaps the most widely accepted opinion as to the mode of infection is that this occurs by means of food contaminated with soil containing the embryos. Further, in ankylostomiasis earth-eating is not an uncommon phenomenon, and reinfection of the host will thereby be facilitated. The drinking of polluted water is, however, regarded by some as of great etiological importance, but there are a number of facts which throw doubt on this being the common mode of infection, one being that, according to Giles, the development of the embryos is retarded or even entirely inhibited by their immersion in water, whilst Looss (1901) states that the embryos do not float in water, but soon sink. A third view as to the mode of infection is based on the experiments of Looss (1901), which show that the embryos can rapidly enter the skin through the hair follicles. He himself claims to have been infected in this manner, and considers infection through the skin to be the usual mode of infection of, at any rate, the field-labourers in Egypt, of tunnel-workers, and of all those whose occupation necessitates their skin being in more or less constant contact with wet and contaminated soil.

Persons in normal health are frequently the hosts of this parasite. Thus it is calculated that 72 per cent of persons in some parts of India harbour this parasite and remain in good health (Fearnside, 1900), and that at least half of the poorer inhabitants of Burmah possess this parasite (Baker, 1900). It is therefore evident that every individual who harbours this parasite cannot, strictly speaking, be said to be affected with ankylostomiasis. Hence this term must be reserved to denote the morbid condition resulting from the presence of the worms. The minimum number of the parasites requisite to cause ankylostomiasis is variously estimated at from 50 to 500, whilst in



many instances enormous numbers are present. The parasite withdraws blood from the host by suction, and this loss of blood is regarded by some as the cause of one of the most prominent features in ankylostomiasis, namely, the severe anæmia; though many suppose that the anæmia is due to some toxic substance excreted by the parasites, or again, to absorption of intestinal toxins through the lesions in the mucous membrane. In the initial stages there is gastro-intestinal disturbance, with progressive loss of weight; anæmia develops rapidly or slowly, and in time becomes profound, and is associated with palpitation, vertigo, and other symptoms of severe anæmia. The red blood corpuscles are reduced by about two-thirds, and the colour index is reduced to about one-half of the normal. There is at times a moderate fall in the total number of leucocytes, but the eosinophile leucocytes are increased. In the later stages there is wasting, cardiac weakness, and dropsy. The diagnosis is readily made by the elimination of other possible causes of anæmia, and by the detection of the eggs in the stools. Post-mortem, the upper part of the small intestine shows, besides the parasites, chronic catarrh, small areas of congestion, hæmorrhage, erosion, and even ulceration of the mucous membrane, and sometimes there are dilatation of the stomach, cardiac hypertrophy and dilatation, and various secondary phenomena.

*Prophylaxis*.—In countries where ankylostomiasis is endemic much may be expected from improved sanitation. In many parts of Europe in which there have been outbreaks of “miners’ anæmia,” as the disease is often termed, good results have been obtained by the suitable erection of latrines, by their regular disinfection with chloride of lime, and by the instruction of miners and others as to the nature and mode of propagation of the disease. In this country, foreigners, and especially Italians, should not be permitted to work in mines until microscopic examination has shown them to be free of the parasite.

In the *treatment* of the disease thymol is the best drug. After a dose of castor oil, 30-60 grains of thymol should be given in the morning to the fasting patient, and this is followed by another dose of castor oil in the evening. Manson recommends that 30 grains of thymol be given hourly for four times. The drug as a rule effects a cure, although the experience of many proves it to be at times inefficacious. In such cases santonin and filix mas may be tried. The preparations of iron are useful after expulsion of the parasites.

Other species of the same genus as *Ankylostoma duodenale* are seen in some of the lower animals. The most important is *Uncinaria trigonocephala*, found in the small intestine and cæcum of the dog and fox. It causes severe and often fatal anæmia in packs of hounds, but is not transferable to man.

The only other representative of the Strongylidæ to which reference can be made is *Sclerostoma equinum*; Syn.: *Strongylus armatus*. It is a common parasite. The adults inhabit the cæcum and upper part of the colon of the Equidæ. The eggs are passed from the bowel, and after the embryos have been hatched out and ingested by the horse, they are chiefly found causing aneurisms of the mesenteric vessels. The thrombi often break up to form simple or septic emboli.

ASCARIDÆ have three oral papillæ, one dorsal and two ventral. There are two genera: *Ascaris* and *Oxyuris*.

*Ascaris lumbricoides* (Linné, 1758); the common round worm. In general form it somewhat resembles the common earth-worm, being cylindrical, pointed at both ends, and of a pinkish-yellow or greyish-yellow colour. It inhabits the upper portions of the small intestine. The male measures



15-25 cm. in length, 0.3 cm. in diameter, the caudal end being ventrally curved and bearing two spicules. The female is larger, and may be as much as 40 cm. long and 0.5 cm. broad. The ovaries are convoluted, and about ten times the length of the female worm. The vulva is at the junction of the middle and anterior thirds of the body. The eggs are oval, about 50-70  $\mu$  long and 40-55  $\mu$  broad, reddish-brown, and have a thick shell, outside which there is often a clear, irregular, albuminous sheath. The eggs are present in great numbers in the faeces, and if they be exposed to moisture after having been expelled from the host, an embryo is slowly or rapidly developed within each egg according to the prevailing temperature, but usually in five or six weeks. Infection occurs, without there being any intermediate host, by means of contaminated drinking water, or by the transference to the mouth of such eggs as contain embryos by the hands after they have been in contact with contaminated soil, and in about ten or twelve weeks fresh ova are being discharged into the intestinal canal of the host.

*Ascaris lumbricoides* is found in all parts of the world, and is one of the commonest parasites. It is particularly common during childhood and puberty, and each host harbours one, two, or possibly hundreds of the worms. The disturbance resulting from intestinal ascarides is as a rule proportionate to their numbers. If there are more than one or two, they usually cause catarrhal inflammation, with diarrhoea, and not infrequently small punctate wounds of the mucous membrane. In rare instances, when there are many worms, they have been known to be coiled up into a mass sufficiently large to cause intestinal obstruction. The ascaris, however, does not always remain confined within the intestine, for it exhibits a marked propensity to migrate. It frequently wanders to the stomach, whence it may be vomited, to the oesophagus, pharynx, mouth, trachea, lungs, nasal cavity, accessory sinuses of the nose, or bile-ducts. The intestinal ascaris may be expected to have numerous intestinal bacteria on its surface, and to transport them into whatever part of the body it migrates. Demateis (1900) also shows that there are both saprophytic and pathogenetic bacteria, bacterium coli, bacillus acidi lactici, streptococci, and others

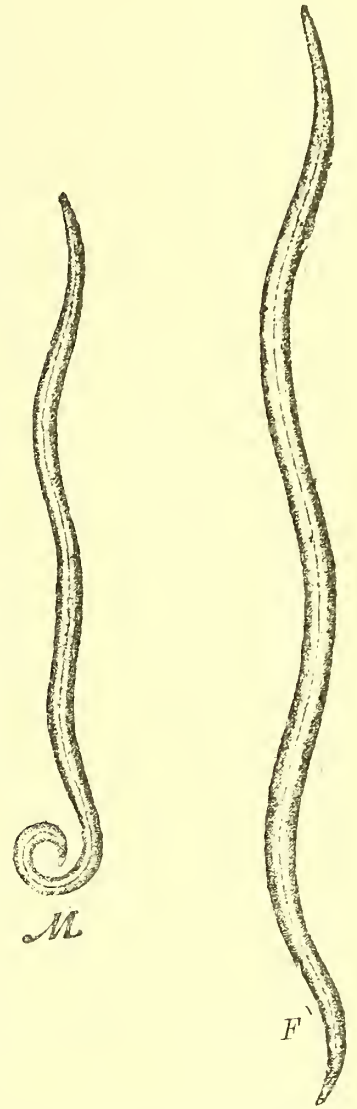


FIG. 24.—*Ascaris lumbricoides*: M, male; F, female; half nat. size. (After Thoma.)

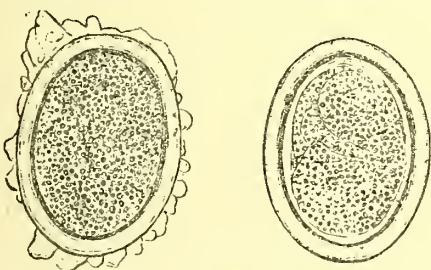


FIG. 25.—Eggs of *Ascaris lumbricoides*. (After Mosler and Peiper.)

in the intestine of ascarides. These facts explain why it is that when the worms wander into the bile-ducts they cause hepatic abscesses, and when they migrate to the lungs cause pulmonary abscess or gangrene. The worms may also migrate from the alimentary canal through the anus, and may then wander into the vagina. Again, the worms have been known to perforate the intestinal wall and cause a localised abscess or peritonitis, and may also migrate into and perforate through the wall of the vermiform appendix causing appendicitis. Perforation of the intestine is fortunately, however, a relatively rare occurrence. There are some authorities who do not believe



that ascarides are ever able to penetrate through the healthy intestinal wall, although admitting that the worms may penetrate through at situations where there are ulcers of tubercular, typhoid, or other nature.

In many patients there are nervous symptoms which used to be commonly regarded as reflex phenomena, but which are now often referred to some toxic substance which the worm naturally possesses or excretes as a product of its metabolism. Of such phenomena, which are most frequently seen in children, nervous irritability, pruritus and picking of the nose, grinding of the teeth, strabismus, dilatation of the pupils, headache, muscular twitchings, choreic movements, convulsions, and urticaria may be mentioned. It is usually difficult to determine to what extent such symptoms as these are due to the presence of intestinal parasites. In many cases they are quite independent of any such cause, but the phenomena entirely cease, in not a few cases, after expulsion of all the ascarides.

Unless the parasites are passed spontaneously the diagnosis is to be determined by detection of the characteristic ova on microscopic examination of the fæces. Only a small quantity need be examined, for if there are any ova at all they are numerous. A common and useful method is the examination of the material filling the eye of an ordinary metal catheter, after it has been passed a short distance into the rectum.

*Treatment* is commenced only after detection of ascarides or their ova. Santonin is the most reliable drug. It is most suitably administered in powder form, in castor oil, or in the case of infants, in olive oil. When santonin is given to a fasting patient, it is a more efficacious anthelmintic, but is then generally regarded as more liable to induce yellow vision (xanthopsia), vertigo, vomiting, convulsions, or other toxic effects. The drug should be given on two or three successive nights, and followed by castor oil each morning. The worms are often, but not invariably, dead when expelled. Turpentine, thymol, and naphthalin are recommended by some as substitutes for santonin. Attention should be paid to the patient's diet; carbohydrates and all articles containing sugar should be prohibited as far as possible, milk, fish, and meat forming the chief articles of diet for a few days.

Various species of ascaris are parasitic in the lower animals. One of the most frequent is *Ascaris canis*; Syn.: *A. mystax*, which occurs chiefly in the cat and dog. It is claimed to have been found in one or two instances in the human subject.

*Oxyuris vermicularis* (Linné, 1767); the thread-worm.—The male is 3-5 mm. long, the caudal end is obtuse and ventrally curved, and bears one spicule; the female is 10 mm. long, the posterior end pointed.

FIG. 26.—*Oxyuris vermicularis*, male and female. (After Gerhard-Seifert.)

Both are white in colour. The female is oviparous, the eggs being oval, 50  $\mu$  long and about 20  $\mu$  broad, and the shell dense, though somewhat thin. The embryo within the shell is often developed whilst the egg is still in the uterus, but if not, is rapidly developed in the presence of warmth, for example, in the fæces. The life-cycle is completed without a change of host. Infection occurs by the ingestion of eggs along with contaminated water, uncooked vegetables or fruit. The shell is then digested and the embryo, after escaping, develops into the adult worm.

This parasite is found in all countries, and is probably more common than any other entozoon, being specially frequent in children. Its habitat is the lumen of the large intestine, more especially the cæcum, sigmoid



flexure of the colon, and rectum, though the worms are also found in the ileum. M. A. Ruffer (1901) in one case found the eggs in the wall of the rectum, and he suggests that the female may frequently penetrate the mucous membrane. The worms frequently migrate from the rectum, chiefly on to the skin in the anal region, and may thus reach the vulva. The presence of oxyurides in the rectum and likewise their migration cause great pruritus, and the scratching to relieve this is often followed by the adhesion of eggs to the fingers and reinfection of the host.

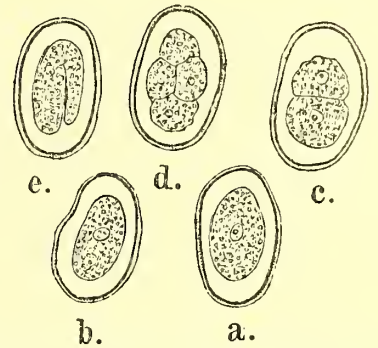


FIG. 27.—Eggs of *Oxyuris vermicularis*. (After Mosler and Peiper.)

In some instances the oxyurides cause no symptoms, and are detected accidentally. In the great majority of patients, however, irritation and pruritus in the anal region are the chief symptoms; both are intensified at night-time, because the oxyurides then tend to migrate from the rectum, and the patient's sleep is often very markedly disturbed. Secondary lesions, the results of scratching, are not uncommon in the anal and perineal regions. Other common symptoms are intestinal catarrh, with diarrhoea, tenesmus, prolapsus recti, and enuresis. In female patients the worms not infrequently migrate to the vulva and vagina, causing vulvo-vaginitis and in some cases leading to masturbation. In some patients indirect nervous symptoms, resembling those produced by *Ascaris lumbricoides*, may be observed.

In every patient in whom there is nocturnal pruritus and oxyurides should be suspected. If they be present, they are easily seen like small, white, actively motile threads on inspection of the anus or dejecta. If the worms cannot be detected by the naked eye, microscopic search for the eggs is of but little value.

Treatment should not be commenced until the presence of the worms is ascertained. They are not situated solely in the rectum, hence it is evident that treatment per rectum alone will seldom, if ever, bring about the expulsion of all. On seven to ten consecutive days, after the bowels have moved, a rectal injection of a strong solution of common salt or infusion of quassia is administered, whilst the hips are elevated. The patient should be directed to retain the injection as long as possible. Castor oil, jalap, calomel, or, in an intractable case, santonin, are to be given every second or third day for a week or ten days. Any antiseptic ointment is useful in lessening the pruritus, and, as in infection with ascarides, an appropriate diet should be given. Notwithstanding energetic treatment it is often months before the patient is rid of all the worms.

**ACANTHOCEPHALI.**—The Echinorhynchi are cylindrical unsegmented worms, without an alimentary canal, but having a protractile proboscis anteriorly armed with hooks. The adults inhabit the alimentary canal of vertebrates, the larvæ being encysted in the visceral cavity of crustacea or insects. There are about two hundred different species of these worms, parasitic in various vertebrate hosts, but Echinorhynchi are very exceptionally found in man. That they may develop in the human intestine, however, was shown by Calandruccio (1888), who swallowed larvæ of *E. moniliformis*, and from whom fifty-three adult Echinorhynchi were expelled thirty-nine days later.

*Echinorhynchus gigas* (Goeze, 1782).—In some parts of the world, though not in this country, it is a common parasite in the small intestine of the pig, and is frequently present in large numbers. The length of the male is



6-9 cm., that of the female 20-35 cm. In a few doubtful instances the worm has been recorded in man.

*Echinorhynchus hominis*, found by Lambl in 1859 in the intestine of a boy in Prague. It was 5·6 mm. long.

### ARTHROPODS

The parasitic Arthropods are all included in two classes and in four orders:—

I. ARACHNOIDEA: (a) *Acarina*, (b) *Linguatulida*.

II. INSECTA: (c) *Rynchota*, (d) *Diptera*.

(a) *Acarina* (Mites).—The head, thorax, and abdomen are all fused in one. Anteriorly, there is a rostrum adapted for the purpose of biting, sucking, or adhesion, there are four pairs of articulated legs bearing at the distal end hairs, claws, or suckers. The sexes are separate. The young forms have three pairs of legs, *i.e.* are hexapod.

*Leptus autumnalis* (Shaw, 1790).—It is a hexapod young form, not exceeding 0·5 mm. in length, of a reddish colour, is found on grasses and bushes in summer time, and often attacks man and other animals, sucking blood and causing cutaneous inflammation.

*Pediculoides ventricosus* (Newport, 1850) is 0·1-0·2 mm. in length, is usually parasitic in the larvæ of insects, and has been known to transfer itself to the skin of persons handling grain.

*Argas reflexus*, *Dermanyssus avium*, and other avian parasites, *Ixodes ricinus* of the dog, and other Acari, are occasional and accidental parasites on the skin of man.

*Sarcoptes scabiei* (Linné, 1748); Syn.: *Acarus scabiei* (Linné, 1748).—The Sarcoptes of different animals are merely varieties of one species. *Sarcoptes scabiei*, var. *hominis*:—the body is slightly oval, and when observed from the dorsal aspect only the two anterior pairs of legs are visible. The male is 0·2-0·3 mm. long, and has suckers on the first, second, and fourth pairs of legs. The female is 0·3-0·4 mm. long, has suckers only on the first and second pairs of legs, and inhabits burrows in the epidermis, in which she lays her oval eggs. For further information, *vide* "Scabies."

*Sarcoptes minor* (Fürstenberg, 1861).—There are two varieties (*S. min. cati* and *S. min. cuniculi*). If accidentally transferred to man it causes a slight transient skin eruption.

Parasites of the genera *Psoroptes* and *Symbiotes*, which are of great frequency and importance in the domestic mammals, are not transferable to man.

*Demodex folliculorum* (Simon, 1842); Syn.: *Acarus folliculorum* (Simon, 1842); etc.—The body is elongated. The cephalo-thorax has four pairs of legs, the abdomen is conical and transversely striated. The length of the variety found in man is 0·3-0·4 mm. The animals live as saprophytes in the sebaceous glands, although they sometimes block the ducts and cause comedones. The variety found in the dog is of smaller size, and is the cause of follicular mange.

(b) *Linguatulida*.—The body is elongated and vermiform; the head, thorax, and abdomen are fused together; the sexes are separate, and the adults inhabit the nasal cavities or lungs of mammals or reptiles.

*Linguatula caprina* (Abildgaard, 1789); Syn.: *Linguatula taenioides* (Rudolphi, 1810); etc.—The male is about 2 cm. long, whitish in colour; the female is 8-13 cm. in length, and yellowish. The adults live in the nasal cavities or frontal sinuses of various mammals, and occasionally of



man, causing severe inflammation. The eggs pass from the host with the nasal discharge, and if they are subsequently swallowed with water or vegetables by some other animal, the young form, or larva, escapes from the egg, becomes encysted in the liver, passing through a second larval stage, in which it is known as *Pentastomum denticulatum*, and is 4-6 mm. long. In this state it afterwards wanders from the liver either to the nasal cavities, where it develops directly into the adult form, or wanders to the lungs or intestine, and is thence discharged from the intermediate host, and if it subsequently gain access to the nasal cavity of another host, it then develops into the adult *Linguatula*. The adult has very rarely been observed in man, but statistics show that the presence of one or two examples of the larva is not very uncommon in the human liver in Germany, Austria, Russia, and Switzerland. The larva does not cause evident symptoms, and is only detected post-mortem.

*Porocephalus constrictus* (v. Siebold, 1852); Syn.: *Pentastomum constrictum* (v. Siebold, 1852).—Only the larva is known. It is 13 mm. long, and has in a few cases, and mainly in Egypt, been found in the human liver.

(c) *Rynchota*, or *Hemiptera*, are all ectozoa. The four chief species are: *Pediculus capitis*, *Pediculus vestimentorum*, *Pediculus pubis* (*Phthirius inguinalis*), and *Cimex lectularius* (the bed bug). Vide "Pediculosis."

(d) *Diptera*. Vide article, "Myiasis." All the adult parasitic *Diptera* are ectozoa, obtaining nourishment from the skin of their host. Such are *Pulex irritans* (the common flea), *Sarcopsylla penetrans* (sand flea), found in Central and South America, and on the West Coast of Africa, and other stinging and blood-sucking insects. Lastly, the larvæ of *Diptera* may be entozoa, living chiefly within the skin, the nasal cavities, or alimentary canal of their host, and causing the condition termed Myiasis.

LITERATURE.—The literature on parasites and parasitic diseases is very extensive. Of many standard works, the following may be consulted:—T. SPENCER COBBOLD. *Parasites; a Treatise on the Entozoa of Man and Animals*. London, 1879.—R. LEUCKART. *Die Parasiten des Menschen u. d. von ihnen herrührenden Krankheiten*. 2 Auflage. Leipzig, 1879-1901. A portion of the work has been translated into English by W. E. Hoyle, and is styled *The Parasites of Man*. Edinburgh, 1886.—L. G. NEUMANN. *A Treatise on the Parasites and Parasitic Diseases of the Domesticated Animals*. Translated by G. Fleming. London, 1892.—F. MOSLER u. E. PEIPER. *Thierische Parasiten*, Nothnagel's *Spec. Pathol. u. Therap.* vol. vi. Wien, 1894.—M. BRAUN. *Die thierischen Parasiten des Menschen*. Würzburg, 1895.—R. BLANCHARD. Art. in Bouchard's *Traité de pathologie générale*, vol. ii. Paris, 1896.—The most important original contributions on the subject are contained, or abstracted, in *Centralb. f. Bakt., Parasitenkunde u. Infectns.*, Abth. i., and *Arch. d. Parasitol.*

## Parotid Gland, Disorders of.

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SOME knowledge of the anatomical relations of the parotid gland is necessary, as its disorders fall naturally under the three chief headings of injury, inflammation, and new growths, each of which, from the position of the gland, may involve structures of great importance. A brief description of the anatomy of the parotid, so far as it is useful to the pathology, will form, therefore, a fitting introduction to this article.



## ANATOMY

The parotid glands are the largest of the salivary glands. They lie on either side of the face, and their anatomical relations are similar. Each gland lies below and in front of the ear, stretching forwards on the outer surface of the masseter muscle, where there is often a small and detached portion, *the socia parotidis*. The gland is enclosed in a fossa of prismatic shape, bounded by two layers of fascia forming the parotid sheath. The inner surface of the gland frequently extends very deeply as the pharyngeal prolongation which passes in front of and behind the styloid process towards the internal carotid artery and the internal jugular vein. It thus passes into the subparotidean space, and along the lateral wall of the pharynx, to which it is sometimes attached, and in so doing it is in relation with the deep part of the glenoid cavity. The anterior surface of the gland is grooved externally by the posterior border of the ramus of the jaw, and passes forwards between the two pterygoid muscles, whilst the posterior surface rests upon the external auditory meatus, the mastoid process, the sterno-mastoid muscles, and the posterior belly of the digastric. The deep surface of the gland has a very close relation to such important structures as the internal carotid artery, the internal jugular vein, the vagus, the glosso-pharyngeal, spinal accessory, and hypoglossal nerves. It lies also sufficiently close to the foramen rotundum for pus to pass from the gland into the skull.

The gland is traversed by the external carotid artery, which divides opposite the neck of the condyle of the lower jaw into the temporal and internal maxillary arteries. The transverse facial branch of the temporal passes out of the upper part of the gland at its anterior border. The internal maxillary and temporal veins, forming the origin of the external jugular, and a large branch connecting the external jugular vein with the internal jugular, also pass through the gland superficially to the arteries.

The facial nerve runs forwards in the substance of the parotid gland, crossing the external carotid artery, and dividing behind the ramus of the jaw into temporo-facial and cervico-facial branches which form the *pes anserinus*. The direction of the nerve is forwards and downwards, its situation the lower third of the gland. The auriculo-temporal branch of the inferior maxillary division of the fifth nerve runs upwards with the temporal artery under cover of the parotid, and joins the facial nerve by two branches behind the neck of the condyle. The gland is also supplied by branches of the great auricular nerve and by sympathetic filaments.

The duct known as Stensen's, or Steno's duct, emerges from the anterior border of the gland at the junction of the upper with the lower two-thirds. It passes over the masseter about a finger's breadth below the zygoma, or, more accurately, along a line drawn from the upper part of the lobule of the ear to the ala of the nose. The duct receives the secretion of the *socia parotidis*, and at the anterior margin of the masseter it dips through the fat of the cheek and the buccinator muscle. It then runs obliquely through the mucous membrane of the mouth, and opens at some point in a line drawn along the inside of the cheek from the second upper bicuspid to the second molar tooth. The exact point varies in different persons, but the opening is generally marked by a papilla, and its presence can be demonstrated by the secretion of saliva, which takes place when the patient is made to suck a piece of lemon or other sialogogue. The transverse facial



artery lies parallel to the duct upon the masseter muscle, and at some distance above it. A main branch of the facial nerve crosses the duct from above downwards as it lies upon the masseter.

The parotid gland is enclosed in a double layer of fascia which forms the parotid sheath, and is derived from the deep cervical fascia. One layer of the fascia is attached to the parotid fossa, the other is intimately blended with the substance of the gland. The parotid sheath, however, is not absolutely continuous, for it is pierced by the different structures which enter and leave the gland, and it is incomplete behind between the styloid process and the external pterygoid muscle. The parotid sheath, too, varies greatly in strength at different parts. It is most dense over the front of the gland, and least dense between the digastric and stylo-hyoid muscles. The deeper layer of the capsule is so intimately adherent to the parotid by its interlobular prolongations that it is almost impossible to remove the gland by the intracapsular method. The outer layer of the capsule, on the other hand, is much less adherent to the parotid fossa, though its adherence varies at different parts; thus it is easily separated subcutaneously, over the masseter, near the stylo-hyoid and digastric muscles, in the submaxillary region and behind the jaw, whilst it is very firmly attached to the neighbouring structures along the anterior border of the sterno-mastoid muscle and at the upper part of the fossa. It is easier, therefore, to remove the gland by working behind the two layers of the fascia than by exposing the glandular tissue.

Two to six small lymphatic glands lie more or less embedded in the substance of the parotid gland—the more superficial lie beneath the parotid fascia, immediately in front of the tragus; the remainder are situated more deeply. These glands receive the superficial lymphatic vessels from the upper part of the side of the face and the lower eyelid; the deeper lymphatics from these parts emptying themselves into the lymphatic glands beneath the lower jaw.

#### INJURIES

Wounds of the parotid gland itself are not of very great importance unless one of the vessels or nerves running in its substance chance to be injured, but wounds of the duct may lead to a very troublesome salivary fistula. The gland is most often wounded from the cheek, but there are a few cases on record where the injury has been associated with a wound of the mouth or pharynx. The first indication of serious trouble in connection with a wound of the parotid is free hæmorrhage, and the surgeon must decide at once whether it is venous or arterial. Venous hæmorrhage, even from the large trunks running through the parotid, can be stopped by pressure or judicious plugging of the wound; severe arterial bleeding must be treated by operation. The direction and depth of the wound may afford some indication as to the vessel wounded, whether it is the vertebral, the internal carotid, the external carotid or some of its branches. If possible the bleeding points should be secured in the wound itself, but if this is impracticable, the vertebral or the external carotid should be ligatured, and if necessary a temporary ligature should be placed round the common carotid. When facial paralysis follows a wound of the gland, it becomes a question whether or not an attempt should be made to unite the divided ends of the portio dura. I should be inclined, for my own part, to leave them alone. Extra pains must always be taken in a wound of the parotid gland to ensure union by first intention. The edges of the incision should be accurately united, and the parts should be kept



at rest for the first five days by forbidding the patient to talk, and by the administration of such food as will render mastication unnecessary.

Stensen's duct is more often wounded by vertical cuts than by those which run parallel to its course. It is more often divided on the masseter, where it is comparatively fixed, than on the buccinator, where it is more movable. It is said that the duct has occasionally been ruptured without any external wound, but the recorded case is capable of another explanation. The open ends of the duct can easily be seen when the cheek is completely divided, but in the more usual incised wounds the nature of the injury is overlooked until the continuous flow of a clear fluid, poured out in increased quantity when the patient eats, enables a diagnosis to be made. A salivary fistula is the common result of the accident, but there is no doubt that such wounds occasionally heal without the formation of a fistula, and it occasionally happens that the proximal end of the duct becomes obliterated, and atrophy of the gland results. Stenosis of the injured duct sometimes leads to the formation of a cyst filled with saliva, due to a yielding of the duct wall at the seat of injury, in much the same way as a traumatic aneurysm is produced. The immediate treatment consists in carefully suturing the parts if the wound is partial; or if it extends completely through the cheek, a fine drainage tube may be introduced from the buccal surface through both ends of the duct, whilst the external surface of the wound is carefully sutured.

#### INFLAMMATORY CONDITIONS

The parotid gland is liable to all the ordinary forms of inflammation, acute and chronic, primary and secondary. The most common and best known form of acute infective parotitis is "Mumps," which has been considered already in a separate article (vol. viii., p. 171). A chronic inflammation of the gland sometimes begins as a catarrh of Stensen's duct, or of the duct of the socia parotidis, and such an inflammation is described by German pathologists under the term *sialodochitis fibrinosa*, whilst the French speak of it as an acute ranula. Mr. Raymond Johnson has called attention to an interesting form which, as only a few lobules of the gland are inflamed, is comparable with the localised inflammations sometimes occurring in the mammary gland.

Secondary inflammations of the parotid gland afford material for a very interesting study, as they cannot always be explained by the infective theory of disease. The relationship which exists between the parotid gland and the generative organs has been long known, in consequence of the metastatic inflammation which is often seen in mumps, where orchitis and mastitis, and more doubtfully ovarian inflammation, are recognised complications. A simple injury of the testis, and the introduction of a pessary into the vagina, have been followed by an inflammation of the parotid gland. Some surgeons believe that parotitis is especially likely to occur after operations on the female generative organs, even when there is no indication of general pyæmia. Parotitis is an occasional complication of typhus, relapsing, and enteric fever; more rarely it is a complication of gout. Recurrent swelling of both parotid glands has been noticed in some cases of xerostomia or dry mouth, and these cases give a clue to the cause of the inflammation in typhoid fever, for the irritation due to the dryness of the mouth extends from the buccal mucous membrane along Stensen's duct to the glandular alveoli. Mr. Stephen Paget has carefully considered the relationship of parotitis to pelvic and abdominal lesions. He believes that



cases of parotitis associated with injuries to these parts form a group by themselves, and are not as a rule accompanied by signs of septicæmia or pyæmia.

Suppurative parotitis, or parotid bubo, is perhaps the most common and best known form of parotid inflammation next to mumps. It occurs in weakly subjects after childbirth, and as a sequel of erysipelas, typhoid fever, cholera, syphilis, diphtheria, and smallpox. It is a result of mercurial poisoning and of any acute infective inflammation of the gland, or it may start from a boil, from inflammation of the temporo-maxillary joint, or in an osteitis of the lower jaw. The onset of the attack is usually marked by the ordinary symptoms of an acute inflammation, though the commencement may be masked when the patient is suffering from a serious illness. The parotid region is swollen, the swelling being hard at first, but becoming œdematous and diffusely red at a later period. Every movement of the jaw is painful, and the patient can scarcely open his mouth, and has difficulty in swallowing. The swelling attains its maximum about the third or fourth day, and is elastic, but does not fluctuate; it may then undergo resolution, but much more often it suppurates; in rare cases the swelling increases rapidly, and the gland becomes gangrenous. The suppuration occurs quickly, and as the pus is formed beneath the strong capsule of the gland, it burrows in many directions before it points beneath the skin. It may pass downwards into the chest, upwards into the cavity of the skull, backwards along the pharynx, into the middle ear, or into the articulation of the lower jaw. Serious hæmorrhage may take place from ulceration of the large vessels running through the gland, or the facial, jugular, and even the cavernous sinus may become thrombosed. Destruction of the facial nerve may lead to an incurable facial paralysis.

The treatment is either prophylactic or curative. The prophylactic treatment consists in carefully cleansing the mouth in all cases where it is likely that an infective inflammation may spread to the salivary glands. The curative treatment requires that the presence of pus should be recognised as soon as possible, and that means should be taken immediately to permit of its escape, and to prevent its accumulation under pressure. The pus is evacuated by drawing the gland well forwards, and then making an incision parallel with the main branches of the facial nerve. The scalpel should not be entered behind a line drawn from the front of the condyle to the angle of the lower jaw, lest the external carotid artery be injured, and it should be laid aside altogether as soon as the capsule of the gland has been divided. The operation is completed by passing a pair of dressing forceps into the gland until pus escapes. The incision through the skin, and the opening into the gland, must be of sufficient size to allow the pus to escape freely, and a drainage tube should be inserted to prevent any reaccumulation. The whole region must be well fomented with boracic compresses.

Syphilitic and tuberculous inflammations of the parotid have been described, but although they are possible, it seems more likely that the condition described has been due to enlargement of the lymphatic glands which lie in and upon the gland. (See also article "Salivary Glands.")

### TUMOURS

The tumours of the parotid gland have been very carefully studied by many observers, for they offer points of great interest. Like tumours in other parts, they are extrinsic or intrinsic, innocent or malignant, solid and cystic. The extrinsic tumours are those which lie above the capsule of



the gland, and may yet be mistaken for parotid tumours. Sebaceous cysts, dermoids, and enlarged lymphatic glands are the more common forms. Dermoid cysts in the parotid region sometimes present a peculiar modification of their contents, which seems to be almost pure oil instead of the ordinary secretion of the skin.

Nævi, cystic lymphangiomata, lipomata, and lymphadenomata also occur in the cheek over the parotid. These tumours are usually bounded by the capsule of the gland, and are thus extrinsic, but they sometimes involve the substance of the gland itself. The true nature of a parotid nævus may be overlooked, as the skin remains unaffected, but they may be removed without dangerous bleeding if the ordinary precautions be taken to arrest hæmorrhage at the time of the operation. The cystic lymphangiomata sometimes show a tendency to disappear spontaneously, but as they are usually large, unsightly, and occur in children, who may thus be prevented from receiving an ordinary school education, I prefer to remove them.

Single cysts, formed slowly and filled with a clear viscid saliva, are found in adults. The wall of the cyst is lined by cylindrical epithelial cells which become tessellated in process of time. The cyst appears to be a dilatation of one of the smaller branches of Stensen's duct. It may be opened, and the epithelial lining destroyed by the application of a solution of zinc chloride (40 grains to the ounce), or pure carbolic acid, which probably occludes the undilated portion of the duct at the same time. Simple puncture gives unsatisfactory results, as it may be followed by the formation of a salivary fistula. The cyst may be dissected out of the gland, but there is some risk of injury to the facial nerve. A second form of cyst, found in connection with the parotid gland, is a dilatation of Stensen's duct. It is found in glass-blowers as a result of their occupation, and is best left untreated.

Fibromata and myxomata are amongst the rarer innocent tumours of the parotid, but the commonest and best known clinically is the "mixed parotid tumour." These tumours begin to grow in patients between the ages of twenty and thirty. They have no uniform rate of growth, for they sometimes remain stationary for long periods and then increase rapidly; more usually the growth is slow but continuous, until they reach a very large size, as in the case recorded by Mr. Sydney Jones (*Trans. Path. Soc.* vol. xxiii., 1872, p. 263), in which the tumour weighed 3 lbs. 14 oz. They retain a rounded outline and feel knobby. The skin over them is normal, except for some venous congestion, and it is movable upon the tumour. The tumour, too, is often movable upon the underlying tissue, and seems much more superficial than is really the case. At first there are no functional troubles, but at a later period hearing is diminished, and the facial nerve is paralysed by the pressure exercised by the growth. A guarded prognosis should be given about these tumours. They may at any time begin to grow rapidly without apparent cause, but they do not usually recur after removal. The treatment consists in dissecting them out as soon as possible, and if care be taken, neither the vessels nor the nerve need be injured. Histologically they have given rise to much discussion, but they are placed now amongst the endotheliomata. The tumour, on this theory, is derived from a multiplication of the endothelial cells lining the lymphatics and the capillary blood-vessels. The growth of the endothelial cells fills the lymphatic vessels and spaces with a hyaline substance arranged in a tubular manner, and it is this substance which has been called cartilage, though it has only a superficial resemblance to true hyaline cartilage. Some of the endothelial cells may develop into, or, perhaps more



properly, are replaced by fibrous tissue by the same process which gives rise to adhesions in the interior of diseased joints, whilst other cells undergo mucoid degeneration. New blood channels may be formed in the endothelium derived from the blood-vessels, and the tumour may thus possess in parts a nævoid or angiomatous character.

Pure chondromata of the parotid are found occasionally as slowly-growing tumours which contain true hyaline cartilage.

The malignant tumours of the parotid are either sarcomata or carcinomata, but they are not very common, and most of the recorded cases may be referred to the class of endotheliomata. True sarcomata are rare. They are either spindle-celled, solid, or cystic, or they are melanotic. The spindle-celled sarcomata are often circumscribed at first, and do not involve the lymphatic glands. The melanotic sarcomata grow rapidly, invade the neighbouring lymphatic glands, and soon involve the whole parotid.

The carcinomata are either primary, when they begin in the gland itself, or they are secondary to cancer of the pharynx, face, cheek, conjunctiva, or lower jaw. The primary cancers are described as being either scirrhus or encephaloid, the secondary as epitheliomata of the tubular or scaly variety, but it is doubtful whether such a classification can be any longer upheld.

Malignant disease of the parotid in many cases is readily distinguishable from the innocent tumours. In malignant disease the growth is more rapid, and speedily infiltrates the surrounding tissues, so that it is fixed. The lymphatic glands are enlarged, the skin becomes infiltrated, and if the growth is allowed to continue it fungates and bleeds. Facial paralysis occurs early, and the unhappy patient suffers from severe neuralgic pain. Mr. Butlin lays particular emphasis on the facial paralysis, for he says that, if the nature of the tumour is doubtful and the facial paralysis is well marked, he is inclined to consider the tumour as malignant, the paralysis being due to the implication of the nerve in the growth and not to pressure alone. A soft growth, too, is more likely to be malignant than one which is hard. The prognosis of malignant disease of the parotid is grave. Early diagnosis with speedy removal of the tumour gives the patient a slender chance of recovery. The parotid is best separated from its attachments by dissecting it out from above and in front, and afterwards by approaching the fossa behind the jaw, also from above and in front. Each vessel should be clamped in two places before it is divided, and no attempt should be made to save the facial nerve. The operation must be as thorough as possible, for an incomplete removal is often followed by so rapid a growth of the part left behind that the surgeon seems to have done harm rather than good by his operation.

#### FISTULÆ

Parotid fistulæ occur in connection with the gland or the duct. They may follow a wound in the parotid region, the opening of a parotid abscess, tuberculous, or more rarely syphilitic ulceration, inflammation due to the presence of a calculus or suppuration connected with necrosis of the jaw. The orifice of the fistula is generally very small, and the skin around it is healthy. A clear alkaline fluid trickles from it, the quantity increasing during mastication. It is said that this fluid can be distinguished from the fluid flowing from a lymphatic fistula by the addition of a few drops of perchloride of iron, when a blood-red colour is apparent in the case of saliva, owing to the presence of sulphocyanide of potassium. Parotid fistulæ, like fistulæ of other parts of the alimentary tract, usually close spontaneously,



unless there is some obstruction in Stensen's duct. The treatment varies according to the position of the fistula. When the opening is situated over the gland, and there is no stricture of the duct, the edges may be refreshed and united by a silkworm-gut suture. But when the parotid duct is narrowed or occluded more extensive operations are required. The duct may be dilated by passing a catgut bougie from the mouth, the distal portion of the canal may be laid open inside the cheek, or an attempt may be made to form a new duct.

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**Paroxysmal Hæmoglobinuria.** See HÆMOGLOBINURIA.

**Patella.** See KNEE JOINT.

**Pediculosis.**

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THE terms pediculosis and phthiriasis are now used in a general way for the symptoms produced by lice, whether of head, body, or pubes, although when employed without qualification they are taken as applying to the body-louse.

*Manner of Feeding.*—There has been a good deal of discussion as to the manner in which the louse attacks the skin. Schmarda, Kaposi, and others hold that the insect first bites with its mandibles and then sucks up the blood. Schiødte, a Danish naturalist, who investigated the matter, says it has no mandibles or other means of biting, but only a sucking apparatus which it protrudes at pleasure. On this point modern observers have been anticipated by Swammerdam and Leeuwenhoek, who worked out the insect's anatomy. According to Leeuwenhoek, whose plates are worth looking at, the body-louse first protrudes a sheath (proboscis or haustellum) continuous with the gullet. This sheath is provided with a "piercer," which is introduced between the epidermic scales to puncture a blood-vessel lying underneath. The more readily to do this the louse places itself on its head. Blood passes with a swift motion into the body, where it is incessantly driven backwards and forwards, as any one will have observed who has examined a pediculus corporis gorged with blood after a meal. Leeuwenhoek points out that these rapid movements prevent the coagulation of the blood, an occurrence which would be fatal to the animal. Swammerdam and Schiødte state that the louse attacks a sweat-pore. Schiødte showed that the haustellum was provided with four short threads, which by approximation formed the piercing apparatus. In his figure red blood corpuscles are represented *in situ* in the haustellum.

*Classification.*—Zoologically lice are insects (six legs), and classed among the Hemiptera, sub-order Anoplura (Aptera). According to M'Lachlan, some writers do not consider them true insects. They do not undergo metamorphosis, but develop directly from the ovum or nit. This absence of metamorphosis, I think, can be accounted for by the parasitic life of the animal on the surface of the skin. To survive, it is important it should reproduce quickly and in numbers, so that the intermediate stages have been in the long run missed out. Its life on man, too, makes it independent of seasons and difficulties as to food-supply.



*Variation in Colour.*—It is said that lice vary in colour according to the host :—Grey in Europeans, black in negroes, yellowish-brown in the Chinese, and white in Esquimaux. A. Murray of Edinburgh, who examined lice collected in various countries from the different races of man, found they differed not only in colour, but also in the structure of the claws and limbs. Possibly this variation in hue is an instance of protective coloration.

Three kinds of pediculi are recognised as parasitic on man. They are named after the part of the body they infest, viz.:—*Pediculus capitis*, *pediculus corporis*, and *pediculus pubis*. With regard to the last-named, Leach places it in a separate genus, under the name of *Phthirius inguinalis*, a distinction which meets with the approval of Chatin. As to Alt's *pediculus tabescentium*, or distemper louse, it is not a separate species, as it has been identified as the *pediculus corporis* (Claus).

*Anatomy of the Pediculi.*—The chief anatomical points of the three kinds are as follows :—

*Pediculus capitis* (*le pou de tête*, *die Kopflaus*).—The female head-louse is from 2 to 2·5 mm. long, by 1 mm. broad. The male is smaller (about 1·5 mm. long), and it is found in much smaller numbers.

The head of the *pediculus capitis* is triangular.

The female has the last abdominal segment notched at its extremity and on its ventral surface, the vaginal orifice being placed in the latter situation.

The last segment is rounded in the male, and bears the penis on its dorsal surface. In copulation the female is therefore on the back of the male.

The ova or "nits" are deposited on the hairs, to which they are firmly attached by means of a cement substance. They are provided with a lid or operculum at the free end. A number of them may be found on one hair, the lowest nit being the one first deposited and the first to hatch out ; but as a rule there is only one nit on a hair. Aristotle thought nothing was produced from the nits.

The period of incubation is six days, and in some fourteen days after hatching out the animal is fully developed.

*Pediculus corporis* (*le pou du corps*, *die Leiblaus*, *die Kleiderlaus*, *pediculus vestimentorum*, *tailor's louse*).—The body-louse is very like the head-louse, only that it is larger, attaining to 3 mm. in length by 1 to 1·5 mm. in breadth, has a rather more oval head and more powerful legs, provided with larger claws.

The nits may be deposited on the small hairs of the skin as well as in the clothes, a fact mentioned in Rees's *Cyclopædia*, but apparently quite lost sight of, until Allan Jamieson called attention to this important point.

The body-louse is very prolific. Leeuwenhoek calculated, from a quaint experiment on himself, that two females might in eight weeks produce as many as 10,000 descendants.

*Pediculus pubis* (*Phthirius inguinalis*, *Le pou du pubis*, *le morpion*, *die Filzlaus*).—This louse differs very much from the two previous ones, and its popular name, crab-louse, is not at all a bad one. As a whole it is much broader and flatter, with a rounded head, provided with prominent eyes. The thorax and abdomen are not sharply divided off. The anterior pair of legs differ from the two posterior pairs, in that the latter are armed with strongly curved claws, with which the animal firmly anchors itself.

The nits are deposited on the hairs. The young hatch out in a week, and reach maturity in two weeks.

The symptoms, diagnosis, and treatment will now be discussed.

## PEDICULOSIS CAPITIS

*Symptoms.*—The presence of the parasite in the head gives rise to great irritation, which leads to scratching, with the production of excoriations and pustular lesions. The latter are the result of the planting out of the staphylococcus by the nails, leading to *impetigo contagiosa*, which is not limited to the scalp, but may be found scattered about the face and body beyond the base of operations of the pediculus. The occipital region is the part most affected, and here the hair becomes sticky and matted together. The neighbouring glands are often enlarged, tender, and inflamed, as a result of the local pus irritation ; they may even suppurate in bad, neglected cases. This glandular enlargement is usually the symptom upon which the mother of the child insists as the primary one, the pediculi being



considered as the effect rather than the cause in accordance with the old superstition of spontaneous generation. Besides, it is a comforting theory, which absolves the mother from neglect.

When the hair is worn long, scratch marks and pus lesions may be found about the neck and the upper part of the back, although the pediculi are only present in the scalp. It is said they may sometimes be found in the beard.

*Plica polonica* was thought at one time to be a special disease of the hair. It is really very chronic, neglected pediculosis capitis, in which the hair becomes matted together from pus and filth.

*Diagnosis.*—In the case of children with pustular lesions at the back of the head, ten to one it is pediculosis capitis. This can be confirmed by examination, with a lens if necessary, when the nits will be readily seen. They are adherent to the hairs, and can be readily distinguished from epidermic scales. The pediculus itself may be discovered, but the presence of nits is quite sufficient to clinch the matter, and will serve to differentiate the condition from simple impetigo contagiosa. The same applies to adults with irritation about the back of the head.

*Treatment.*—Where it is not imperative to preserve the hair, as in most children, it should be cut off close, thus getting rid of the adherent nits. The crusts must be softened with oil and picked off (the latter is an important direction), and the following ointment smeared on four or five times a day:—Hydrarg. ammon. gr. x., ol. olivæ ℥x., adipis ʒj. In this way the pus cocci and pediculi are destroyed.

Where the hair cannot very well be cut off, in women and some children, it should be sponged with a carbolic acid lotion, 1 in 40, as follows:—A small portion of the hair is taken up with one hand, and a sponge soaked in the lotion drawn along it with the other, so as to thoroughly come into contact with the nits, and so on until the whole of the hair has been dealt with; or the hair may be spread out on a table, the patient sitting on a stool. The nits can then be combed out. Simultaneously the ung. hydrarg. ammon. dil. is used as already described. Acetic acid or vinegar is also good for getting rid of the nits.

Ordinary petroleum lamp oil is very effectual, soaked freely into the head, the above ointment being used for the crusted lesions. The drawbacks of petroleum are its smell and its inflammability. The latter is a very real danger. It should, therefore, never be applied in proximity to a lighted lamp, etc. It is safest to apply it during the daytime. Oily bodies kill lice by obstructing the tracheæ, and thus preventing respiration.

Other remedies are used, but the above treatment properly carried out will cure the condition. *Le mieux est quelque fois l'ennemi du bien.*

#### PEDICULOSIS CORPORIS VEL VESTIMENTORUM

This disease is usually observed in the old, neglected, badly nourished, and cachectic. It is frequently contracted in common lodging-houses by people who have come down in the world; in some cases by sharing an old person's bed.

*Symptoms.*—Although this pediculus inhabits the clothes, it feeds on the skin, and in this way occasions great irritation and much scratching of the parts within reach of the nails.

The pediculus prefers the neighbourhood of the neckband of the under-clothing, probably to get a better supply of air, and it is about the back of the neck and the shoulders that the scratching is most marked. In chronic cases, such as are to be seen in poor-law infirmaries, the constant irritation and scratching leads to pigmentation, which may be very extensive and deep in colour, with thickening of the skin, the so-called "vagabond's



disease," common among tramps. The pruritic sensations are worse at night, and once started, may reflexly involve any part of the body.

Urticaria, pus inoculation, furuncles, and even abscesses, are the complications met with.

Pyrexia has sometimes been observed (as high as  $106.4^{\circ}$ ), and Payne thinks this may be due to a poison inserted by the insect (*see also* "Leeuwenhoek").

*Diagnosis.*—The chief points are the *age* and *appearance* of the patient, and the *distribution* of the scratch-marks about the neck and shoulders. An examination of the under-linen, especially about the neck-band, will reveal the pediculus, unless the vest or shirt is a clean one put on for the occasion.

Tilbury Fox insisted on the importance of the minute hæmorrhagic speck which occurred on the skin as a result of the insect's mode of feeding. Radcliffe-Crocker is inclined to think it is only the excrement of the insect, a very likely thing, considering it feeds on blood, and that when it sucks up its meal the fluid is driven about with such force as to press out the contents of the lower bowel. Dubreuilh and Beille state they have never been able to see this "hæmorrhagic speck."

*Differential Diagnosis.*—This is a matter of some importance, as there is a condition known as pruritus senilis, associated with degenerative changes of the skin, which might lead to difficulty; but in such cases little is to be seen about the skin in the way of scratch marks. To avoid errors, every case should be taken on its merits, and the positive signs of pediculosis relied upon. Pruritus of the body may be a marked symptom in some morbid nerve conditions, such as locomotor ataxy, and also in chronic opium-eaters.

When pigmentation of the skin is present in a marked degree, the question of Addison's disease may arise. Greenhow, Besnier, and Thibierge have seen the mucous membranes pigmented in so-called "vagabond's disease," a point which must be borne in mind, and which favours Besnier's view that the pigmentation is not due to scratching.

Urticaria may be due to a variety of causes, one of which is pediculosis, which would have to be excluded. Where there is doubt, the urine should be examined for albumin and sugar, and the alimentary tract overhauled.

When the scratching has been vigorous and chronic the patient may present pigmentation, superficial scarring, and scattered crusted sores from pus inoculation, so as to suggest syphilis to some minds. The absence of the confirmatory signs of the latter, with positive signs of pediculosis, would clinch the diagnosis. Of course syphilis and parasites may be associated.

In the same way, scabies may be a complication. The itch differs from pediculosis in the localisation and distribution of the scratch-marks; in the former the hands and wrists are usually affected (*vide* "Scabies").

In some instances patients *fancy* they have pediculi. This condition of mind is a pediculophobia, comparable with what is observed in syphilophobes. It is very difficult to convince them that no parasites are present. Exceptionally, the mental attitude may become stereotyped, a pediculosis cerebri, with delusions, and sometimes ideas of persecution. In one case there were auditory hallucinations. The patient heard voices saying he was lousy. These cases generally end in the madhouse. In other instances, again, a patient may have been delivered from the parasites, but will remain convinced that he still has them. These cases also may drift into a pediculosis cerebri condition.



*Treatment.*—The first step is to treat the bed and body clothes, either by baking them in a disinfecting apparatus at 212° F. or over, or where this cannot be carried out, by thoroughly boiling them in the copper.

The patient must next be attended to, and in most cases half-a-dozen alkaline baths (3ii or more of bicarbonate of soda to 30 gallons) to allay the irritability of the skin, soap and water cleanliness, and ung. staphisagriæ to rub into the neck where the neck-band comes into contact, will suffice to effect a cure. In obstinate recurrent conditions the fact that nits are deposited on the lanugo hairs of the skin, as observed by Allan Jamieson, must not be forgotten. Here rubbing in paraffin over the whole body, followed by a warm carbolic acid bath (3ss to 30 gallons), as he recommends, is necessary to get rid of the parasites. The wearing of sulphur bags next to the skin has also been found useful.

Where the patient is much run down from privations general treatment must be added to improve the nutrition and tone.

Pediculophobia and pediculosis cerebri cases are very difficult to deal with. The patients should be listened to, and every effort made to convince them of their mistaken notions. Where delusions and hallucinations exist the prognosis is bad, and such cases are for the alienist.

### PEDICULOSIS PUBIS

This is in most cases the result of impure sexual intercourse, but it may be quite innocently contracted from infected bedding and so forth. This kind of pediculosis is more frequently observed among the better classes than the other two.

*Symptoms.*—Irritation about the pubic region leads to scratching, especially at night. But as the pediculus may extend its operations to the hairs of the abdomen, chest, axillæ, etc., the scratching may be generalized. In a hairy man of about sixty-five I found the insect not only about the pubes, but on the abdomen, chest, shins, and in the axillæ and beard, but not on the scalp. Heisler has recorded the case of a child of fourteen months, in which the scalp was involved, and Trouessart another in an infant of five months. In both cases the disease had been contracted from the nurse. In Heisler's case the eyelashes were also affected. Grindon has observed the pediculus pubis in the scalp in several members of a family.

In some cases a marked papular eruption about the pubes and adjacent parts is present. When irritant applications have been used there may be a severe dermatitis, eczematous in appearance, which may become widespread.

*Maculæ cæruleæ (taches ombrées)*, first described by Morrison, have been observed in the affected regions. They are bluish-grey stains in the skin, about the size of a finger-nail. Duguet has shown that the pigment was contained in a pair of glands situated in the thoracic region opposite the second pair of legs of the insect. The subject has also been investigated by Payne, who has observed pyrexia in connection with the pediculus pubis. Jamieson thinks the stains have some anæsthetic effect. Does the insect anæsthetise the spot it is about to feed on?

*Diagnosis.*—The possibility of pediculosis pubis should always be borne in mind whenever there is itching about the pubes and scratched papules are scattered about this region. If the insect is present a search for it or the nits will be rewarded. As a rule there is little difficulty in coming to a definite conclusion.

Sometimes patients have used irritant ointments, and will seek advice for the dermatitis, saying nothing about the pediculi. The true state of affairs may therefore be masked, and a careful examination necessary.



The *maculæ cæruleæ*, if present, will help, but they require looking for. In hairy persons other parts of the body should be examined.

The patients are usually young men, but neither age nor sex can be excluded.

Oppenheim has recently drawn further attention to a green colouring matter found in the crab-louse. Where the *maculæ cæruleæ* were marked he found that all the mature lice contained this colouring matter in large quantities, the parasite appearing diffusely green. The impregnated females showed the pigment most; young individuals were always devoid of it. There is no corresponding colouring matter in head and body lice.

*Treatment.*—A favourite remedy used to be ung. hydrarg., but it has sometimes led to such severe dermatitis, to say nothing of the possibility of salivation in susceptible subjects, that it is now discarded. The pediculi can be got rid of by less drastic measures. Various ointments may be used, such as: Naphthol  $\beta$  3ss., cret. præp. 3ss., adipis ad 3j.; or balsam. Peruviani, adipis, aa 3ss. Lotions of carbolic acid 1 in 40, or of perchloride of mercury 1 in 500, are useful (the latter carefully applied), the former to destroy the nits, as in pediculosis capitis. Eau de Cologne freely dabbed on will frequently do all that is required.

Of course, where a patient comes with a severe dermatitis, this condition must be treated by means of soothing applications, such as linimentum calaminæ (calaminæ præp. 3iss., zinci oxidi 3j., ol. olivæ, aq. calcis aa 3iv.), or a lead lotion (glycer. plumbi subacetatis 3j., aq. dest. ad 3viiij.).

In ordinary cases, when the insects have been got rid of, a soothing lotion is sometimes necessary, such as calaminæ præp. 3iss., zinci oxidi, 3j. glycerini 3iij., aq. ros. ad 3viiij., dabbed on and allowed to dry.

As in pediculosis corporis, patients, although cured, will get the crab-louse on the brain. In a case under my care, an elderly man, he turned up for some time with bits of dirty wool, scales, etc. carefully wrapped up in paper, under the impression they were pediculi. Even when these objects were placed under the microscope he was but half convinced. Pediculosis cerebri may also occur in such cases, and require careful management. The patient's tales should be listened to, and means taken to convince him of his error, if possible, but it is as a rule a hopeless task.

The pubic hairs should not be cut, for the pressure of the clothes produces great irritation, until the hair has become curled, a fact well exemplified after operations for hernia, etc., when the pubic hairs have been shaved.

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**Peliosis Rheumatica.** See RHEUMATISM.



## Pellagra.

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DEFINITION.—A chronic endemic, non-contagious, cerebro-spinal disease of poverty-stricken peasants, induced by the toxic action of diseased maize. It produces changes in the spinal cord, stomach, and intestines; sometimes ends in dementia; and causes, chiefly in the spring months, an eruption on those parts of the skin exposed to the sun. The name is taken from the Italian "*pelle*," skin, and "*agra*," rough.

SYNONYMS.—*Malatia della miseria*, *Mal del sole*, *Malatia dell' insolato di primavera*, etc. In Egypt it is called "*Qushuf*," meaning chapping or roughness.

*History*.—Pellagra has been known in Spain since 1735, following on the introduction of maize from America about 1700. About 1750 it was found in Italy, about 1820 in France, and towards 1840 in Roumania. Since 1856 it has been epidemic in Corfu, and of later years has been reported from Hungary (1888), Russia, Portugal, Egypt (1893), and other countries.

GEOGRAPHICAL DISTRIBUTION.—In Europe it is known in Northern Spain, Northern Portugal, the south-west of France, Northern and Middle Italy, South Tyrol, parts of Austria-Hungary, Roumania, Bessarabia, and Corfu. From Asia it has not yet been reported, but many medical officers have recognised my photographs as a disease occurring among out-patients in India.

In Africa it is common in Lower Egypt, rare in Upper Egypt, the Red Sea coast, and in Algiers. It has not been reported from South Africa, but I saw two cases there among the coloured lunatics on Robben Island in 1900. In America it exists in Mexico, the original home of maize, but is not known in the United States, where the poorest coloured folk live more luxuriously than the peasants of Italy or Egypt.

It has not been found in Australia yet, but Dr. Neiret has reported it from the island of New Caledonia.

It probably exists in every country where the poorest peasants live habitually on diseased maize.

CAUSES.—Pellagra, analogous to ptomaine poisoning, is a chronic intoxication due to eating damaged maize or Indian corn (*Zea Mays*). Healthy maize, eaten as the chief article of diet, does not produce disease in men or poultry, and, as it contains almost the same percentage of nitrogenous matter as soft wheat and four times as much fatty matter, it stands in a high position as human food, and is more widely cultivated than any other cereal. Townspeople in maize-growing countries who buy their food in the market and vary it with other articles of diet never develop pellagra. In the same way, well-to-do agriculturists and their families and servants eat healthy maize almost daily and never suffer from the disease; but the poorest peasant, who lives from hand to mouth, and either has the habit of buying the cheapest maize in the market, or sells the best of his own crop and keeps the worst cobs for home consumption, becomes in time an easy victim.

Poverty, overwork, and miserable surroundings are all predisposing factors, but are not sufficient in themselves to produce pellagra, which is not an accompaniment of potato disease in Ireland or rice famine in India. As an additional proof that maize is a necessary factor I may mention that



the poorest peasant of Lower Egypt becomes pellagrous because he lives on bad maize, while the equally poor inhabitant of Upper Egypt or the Soudan, living under similar conditions, daily eats millet (*Sorghum vulgare*), and escapes pellagra. Experiments upon dogs, hares, poultry, and men have shown that pellagrous symptoms can be produced by feeding them with damaged maize or with an alcoholic extract prepared from it.

In Italy pellagra was long thought to be hereditary, but its early appearance in that country is probably due to the habit among the peasant women of giving maize "polenta" to children while still at the breast. Both sexes and all ages and races may contract it.

Although we do not yet know what the toxin is, or whether the cause of the intoxication is an epiphyte growing on the cob or in the flour, we know that damp is one of the essentials for producing spoilt maize. It is cut before being ripe, or exposed to rain or dew, or stored away in damp, dark cellars, unprotected in any way from the earth below. Sometimes it is even stored in its sheath, when it is almost impossible for the grain to get thoroughly dry, and sometimes it is grown from seed already diseased or partially eaten by weevils.

In Italy it is thought that it is unwise to grow any species of maize which ripens quickly or ripens during the early autumn months, and it is noticed that pellagra is more apt to be rife after a rainy summer, or after the failure of the crop the year before. It has diminished in Italy since the introduction of improvements in drying and storing the grain.

**SYMPTOMS.**—It is impossible to fix any incubation period, and the course of the disease, if unchecked, must be put down rather vaguely as from five years to fifteen or even twenty. The premonitory symptoms are giddiness, anæmia, failing appetite, general debility, and indefinite pains. When the disease is once established the symptoms can be conveniently divided into those affecting: (1) the skin, (2) digestive system, (3) spinal cord, and (4) brain.

The skin eruptions, although the least important of the various symptoms, have given the disease its name, and have always received an undue amount of attention. They usually appear for the first time during the spring months because the ultra-violet rays of the sun are then especially strong. They die gradually away during the summer, so that only traces can be found in the autumn and winter, but in the spring (and occasionally also in the autumn) they return with redoubled vigour. An erythema not unlike a severe sunburn is first seen, with swelling and tension of the skin for a few days. Later there is, in severe cases, a real dermatitis producing hypertrophy with a gradual loss of elasticity. It is not usually till the second spring that the dermatitis is so extensive as to lead to pronounced exfoliation, but when this is present we have a very characteristic picture.

The epidermis slowly falls off in flakes discoloured gray or brown, leaving a clean surface behind, sometimes in the form of small patches, but more often as striæ. Every year the erythema leaves a little more pigment and thickening behind it, until a chronic atrophy takes place, marking out the place where the eruption has been. The affected skin then looks wrinkled like the hand or neck of a man eighty years old, though the healthy skin in the neighbourhood is still plump and elastic. Early cases complain of burning or tingling in the skin, but later ones only suffer from itching. In advanced cases there is sometimes a curious ichthyotic mottling which seems to be the legacy of many attacks of exfoliating inflammation of the skin, with a deposit of pigment and atrophy of both skin and subcutaneous tissue. The eruption is essentially dry, and bullæ on the extremities are



very rare. The site of the rash is limited only by the patient's clothing. European adults working in the fields get it chiefly on the face, neck, ears, and hands. Those who habitually work with the forearms exposed get it there, those without sleeves have the upper arms affected in addition, and barefooted children are attacked in the dorsum of the feet and legs. The Egyptian peasant wears fewer clothes and works in a stronger sun, and therefore gets more eruption. If he works in the fields wearing nothing



Egyptian girl, æt. 8, showing eruption on face, hands, forearms, feet, and legs.

but a pair of short drawers all the exposed portions of the body will be affected. But even in Egypt the parts earliest attacked with erythema are the hands and forearms, elbows, feet, insteps, legs, and knees, and upper part of the chest. In all countries the rash shows a marked preference for the dorsal aspects of the upper and lower limbs, only creeping round to the flexor side as the case becomes chronic, and never involving the ham, axilla



bend of the elbows, palms, and soles. The dorsal aspect of the middle phalanx of the fingers is affected in long-standing cases, but the ungual phalanx is only rarely involved. The nails and hair are always unaffected. Wherever present the regular symmetry of the eruptions strikes the observer; the erythema, the desquamation or the ichthyotic mottling, extend to exactly the same level, and finish off in the same way as a trophic lesion should do. Asymmetrical clothing will, however, produce erythema occurring differently on the two sides of the body.

2. Examination of the tongue will often help to decide a doubtful diagnosis, for in most of the patients it is more or less denuded of epithelium, while the palate also is bare in some of the worst cases. In early cases it is only the extreme tip and the sides which, on careful examination, show preternatural cleanliness. A few of the worst cases of "bald tongue" show also a painless enlargement of both parotid glands. The tongue is merely an index of the stomach, which is very early affected, as shown by the usual evidences of chronic gastritis, anorexia, epigastric pain, and tenderness, flatulence, and thirst. Vomiting is not present. Constipation or diarrhoea may or may not occur quite early in the attack, but diarrhoea is an almost invariable accompaniment of the last stages. The urine shows no marked abnormality.

Pellagra while untreated invariably causes loss of weight, and eventually produces great emaciation. I have seen patients with diarrhoea and melancholia lose as much as nineteen pounds' weight in hospital in three weeks before death, while the convalescents without diarrhoea have gained from five to thirty pounds. The diarrhoea is believed by Babes to be due to irritation of the sympathetic ganglia and the plexus of Auerbach. There are many reasons for thinking it is tropho-neurotic.

3. If seen in the acute stage an intelligent patient often complains of pains in the back which in severe cases may even compel him to walk with his body arched. On examination, he will have pain on both sides of the dorsal vertebra over the spinal nerves, if gently pressed there with a knuckle. The favourite seat for this tenderness is near the fourth to the ninth dorsal spines, and the tenderness will often be asymmetrical. This symptom invariably disappears after a few weeks' rest, and is not present in quiescent cases. The knee reflex may also be asymmetrical, corresponding with unequal tenderness in the back. At first it is slightly exaggerated, then markedly so, probably associated with irritation of the posterior spinal roots. This is the time of tenderness on both sides of the dorsal and upper lumbar vertebræ, and the abdominal and epigastric reflexes are extremely brisk. Later in the case the knee-jerk becomes dulled, and eventually quite abolished, manifesting a lesion in the posterior nerve roots in the second, third, or fourth lumbar segment. The patient, until he becomes bedridden, does not develop contraction of his lower limbs or paraplegia. He retains control of bladder and rectum, and has no bed sores. The knee-jerk becomes less abnormal while the patient is under treatment if there is an improvement in the other symptoms. Ankle and wrist clonus are very rare, only occurring in a few incurable cases.

Insomnia is an important nerve symptom, coming on very early, and remaining until the patient is given complete rest and good food as in a hospital. There are other nervous phenomena which are rather complications than symptoms of pellagra, such as tonic and clonic convulsions, epileptic attacks, spastic paralysis, atrophy and paralysis of the muscles of the lower limbs, unilateral ptosis, and other eye affections.

4. Among the early brain symptoms are great mental depression, all



thinking and calculating is an effort, and the patient gradually becomes irritable and excitable with others, and stupid and morose when alone. A settled gloom is present on the face after the first year or two, and patients lose all power of smiling and laughing, and some refuse even to try to do so. In advanced cases there are deep transverse furrows of melancholy across their foreheads and their hopeless eyes stare vacantly. During the first or even the second year of pellagra in an individual of average intelligence no definite mental symptoms are noticed. After that he is found to be always sad, decidedly stupid, and unwilling to take any interest in anything beyond his food and sleep. At a later stage he loses his memory and becomes distinctly melancholic, always discontented, craving insistently for food and cigarettes, yet he loses weight in spite of being granted extra food. The sleeplessness now becomes more pronounced, and there are vague feelings about the head; sensation of weight, pressure, emptiness, or pulsation. There is often a vague purposeless restlessness, culminating in a fixed unreasonable desire to get away from the place in which he is.

If the mental symptoms become more evident the patient must be confined in an asylum, for he will require special watching even if he is not suicidal. His delusions when present are insane renderings of his subjective symptoms. The skin irritation causes him to think that he is being burnt, or that his skin is being stretched or peeled off, and that he can escape from his tormentors by plunging into water. His gastric troubles and altered tastes lead him to refuse food and to imagine that every one is poisoning him. Some patients remain in a state of absolute dumbness for months. Those who are not melancholic suffer from the mania of persecution or possession, and the usual ending of mental cases which do not respond to treatment is in secondary dementia, when they become dirty in their habits, forgetful of common decency and most of their past life; they live on until they slowly succumb to diarrhoea or tubercle, perhaps first becoming bedridden from general emaciation and atrophy of their leg muscles. It is interesting to note that some of the chronic pellagrous lunatics in asylums spending several hours a day in the open air develop a new erythema every spring, which is absent at other times of the year. Yet in these asylums maize is not an article of diet.

*Complications.*—There are certain constant accompaniments of pellagra in every country: extreme poverty, insufficient food stuff for constant labour, and a general neglect of all hygienic laws. But, in addition, there are several varying factors which weigh down the unfortunate sufferer. In Europe the pellagrous adults are often addicted to alcohol, and are also victims of uncured syphilis and malaria, after which the tubercle bacillus finds them an easy prey. In Egypt alcohol is not a vice of the peasants, but syphilis and malaria may both occur, and an almost constant accompaniment of pellagra is ankylostomiasis, sometimes joined by bilharziosis; other entozoa and favus of the head may also be present, the patient succumbing eventually to tubercular disease of the lungs.

*Morbid Anatomy.*—At an autopsy there will at once be seen great emaciation and cachexia, generally with marked anæmia. There may be definite exfoliating patches on the parts of the body exposed to the sun during life, or there may be only a little roughness on those parts, but the skin there, if carefully examined, will be found to be atrophied, and there is a general diminution of subcutaneous fat. Microscopically there is sclerosis of the blood-vessels, papillæ, and corium, as well as atrophy of the horny layer.

The muscles, heart, liver, spleen, and kidneys share in the general



atrophy, but the distribution of pigment described by some authors must, I think, be due to concurrent malaria.

The lungs sometimes show tubercular lesions. The stomach reveals no lesion to the eye, but the walls of the intestines are thinner than usual, and show a slight shedding of the superficial layers of the epithelium, with atrophy of the muscular tissue. There is no ulceration of the intestines. Many naked-eye lesions have been reported by various observers as occurring in the brain, but the only constant one is atrophy of the cortex of the convolutions, especially the frontal.

The spinal cord shows no decided change until it is prepared and carefully examined. Tuczek in 1893 found in eight autopsies in Italy that all of them showed symmetrical sclerosis of the columns of Goll. In six cases there was also lateral sclerosis in the dorsal region, and in one case he found cervical anterior sclerosis. Later observers, while confirming the posterior sclerosis, are of opinion that this is perhaps of root origin, and that the increase of the connective tissue in the posterior columns is secondary to degeneration of the roots, for in some cases the sclerosis can be seen to be limited to the degenerate root area. About half the cases lately examined show a parenchymatous neuritis, or a chronic interstitial neuritis of the posterior roots. These results are quite in accordance with the clinical symptoms, and stand out in marked contrast with the appearances of tabes dorsalis.

*Diagnosis.*—A typical case presents no difficulty, because it is the only known disease causing dermatitis on exposed parts of the body, a denuded tongue, sleeplessness, altered knee-jerk, and tenderness on pressure in the dorsal region. The disease occurs in peasants and beggars who live chiefly on maize. Ichthyosis is the only skin disease likely to be confounded with pellagra.

*Prognosis.*—Early cases can be cured by giving a generous diet and excluding maize. Dangerous symptoms are obstinate diarrhoea and insomnia, total loss of knee-jerks, increasing emaciation, suicidal melancholia or dementia, and the concurrence of tubercle.

*Prophylaxis.*—Improve the miserable condition of pellagrous peasants, and teach them the difference between good and bad food. Sound maize flour, damped and kept at a temperature of 77° to 86° F., will remain sweet for more than twenty-four hours, whereas diseased flour will give off a disagreeable odour in less than eight hours. The Government should inspect all markets and prevent the import of sea-damaged maize. Italy since 1890 has provided some of the pellagrous districts with improved means for drying, storing, and baking maize, besides retreats for the care of patients.

*Treatment.*—Fresh air, rest, ablutions, and a liberal diet are the first essentials. Even sound maize should be excluded from the diet for fear of unconsciously adding to the poison. If iron or quinine are not specially called for by anæmia or malaria, strychnia and arsenic will be found the best tonics. Baths and zinc ointment will help the skin eruptions to disappear. Insomnia may require opium or bromides, and the diarrhoea will tax every effort of food and drugs. Since 1889 attempts have been made to find an antitoxic serum by Babes and others.

*LITERATURE.*—References from 1866 to 1899 may be found in SCHEUBE'S *Die Krankheiten der Wärmern Länder*, 2nd ed. 1900.—Recent references are: AGOSTINI. *Manuale di Psichiatria*, 2nd ed. 1900, p. 180.—BABES, v. et SION, v. "Lesions nerveuses dans la Pellagre," *Roumanie médicale*, Nov. 1899.—*Idem*. "Die Pellagra," third section of vol. xxiv. of Nothnagel's *Special Pathology and Therapeutics*. Vienna, 1901.—MANICATIDE, E. "Beiträge zum Studium der ätiologie der Pellagra," *Roumaine médicale*, 17th Aug. 1900.—ROSSI. *Annali di Freniatria*, 1900.—SANDWITH, F. M. "Pellagra in Egypt," *Brit. Jour. Dermatol.* vol. x. —*Idem*. "Three Fatal Cases of Pellagra, with Examination of the Spinal Cords," *Journal of Pathology and Bacteriology*, Autumn 1901.



**Pelvis.****Perineum and Pelvic Floor**

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INTRODUCTORY.—*Structural Anatomy of the Pelvic Floor*

THE most striking characteristics of the female pelvic floor depend upon the fact that it is so constructed as to open during parturition to admit of the passage through it of the foetus. While it has to allow occasional opening for this purpose, the pelvic floor has also to support the abdominal viscera and resist the intra-abdominal pressure throughout life. The arrangements which make parturition possible introduce into the structure of the pelvic floor certain inherent weaknesses, which under certain circumstances are the prime causes of the pathological conditions discussed in the following pages:—

The female pelvic floor may be considered as consisting of two portions: the one which is the more movable being supported by the other, which is the more firmly fixed to the bony structures. The movable portion, which is the inner and anterior portion of the pelvic floor, is connected with the outer and posterior fixed structures by a ring of loose connective tissue which runs round the pelvis. To speak more definitely, the movable portion consists of the bladder, the urethra, and the anterior and posterior walls of the vagina. The uterus, the broad ligaments, and the ovaries and tubes, rest upon these mobile structures, and move with them. The loose tissue by which these organs are connected with the more fixed portions of the pelvic floor extends behind the pubes, along the inner surface of the obturator internus and the upper part of the levator ani, being continued between the posterior wall of the vagina and the anterior wall of the rectum. The use of this ring of loose tissue is not only to render parturition possible, but to allow of the movements of the bladder and rectum, which occur during the filling and emptying of these organs. In prolapse of the uterus, which is, strictly speaking, a form of hernia (sacro-pubic hernia), the whole of the movable portion of the pelvic floor descends, together with the uterus and appendages. Coils of intestine descend in turn and occupy a sac formed by the organs mentioned, lined by peritoneum, and having for outer covering the inverted walls of the vagina.

To obtain a clear idea of the structure of the pelvic floor, it is necessary to consider a sagittal mesial section of the pelvis. In Fig. 1 such a section is figured, the straight lines which are introduced showing the direction in which the intra-abdominal pressure acts upon the various portions of the pelvic floor. Consideration of such a section shows that the vagina is a transverse slit in the pelvic floor, and divides it into two portions—an anterior or pubic and a posterior or sacral portion. The anterior portion is composed of the bladder and urethra, with the anterior wall of the vagina; the posterior portion consists of the posterior vaginal wall, with the rectum, and all the muscular and connective tissue attached to the



sacrum and coccyx, including, of course, the perineum. Fig. 2 shows the appearance of the sacral segment in mesial sagittal section as figured by Hart.

It is thus seen that the division of the pelvic floor into movable and fixed portions, nearly, though not entirely, coincides with the division into pubic and sacral segments. For the posterior vaginal wall forms a part of the movable portion of the pelvic floor, with the anterior vaginal wall, bladder, and urethra; but, on the other hand, it forms a part of the sacral segment, together with the rectum, perineal body, and muscular structures attached to the sacrum and coccyx.

When the uterus contracts during parturition it draws upward all those parts of the movable portion of the pelvic floor which are free to rise, namely, the bladder and the anterior vaginal wall. The posterior vaginal wall is pulled upward certainly, but it is also pushed downward and stretched by the advancing foetal head, together with the rest of the sacral segment of the pelvic floor. Thus the floor opens like a pair of folding doors, one of which is pulled while the other is pushed open. The pubic segment is pulled up, the sacral segment is pushed down, the vaginal cleft being altered from a mere potential slit into a tube four inches wide.

This mechanism is well seen by placing a patient in the genupectoral posture and allowing air to enter the vagina. The

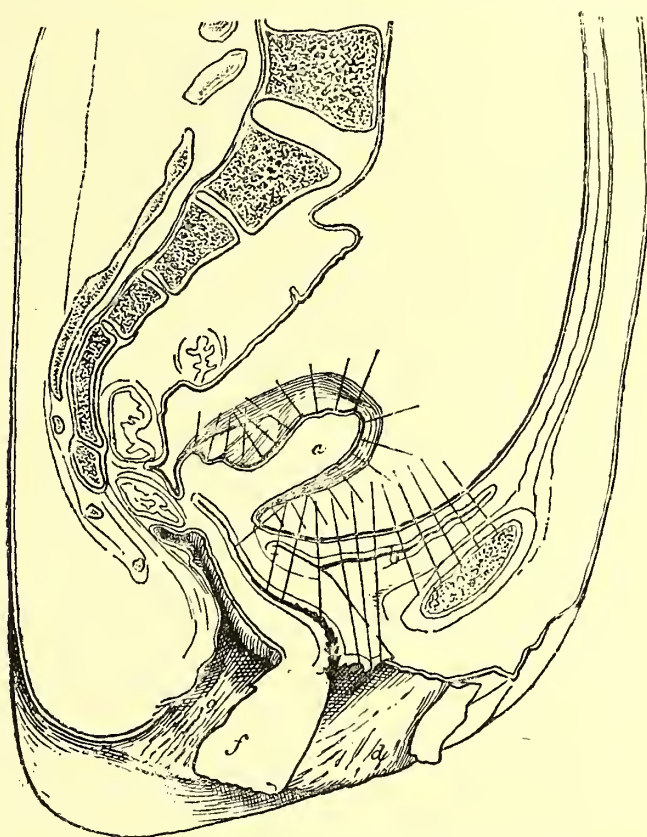


FIG. 1.—Sagittal section of pelvis. *a*, uterus; *b*, bladder; *c*, rectum; *d*, vulva; *e*, symphysis; *f*, perineal body. The lines show the direction ultra abdominal pressure. (Hart and Barbour.)

pubic segment of the pelvic floor then falls away from the more fixed sacral segment, the vagina remaining widely pouched, as long as the pelvic floor is relieved of intra-abdominal pressure by the maintenance of the genupectoral position. A study of the lines of pressure drawn in Fig. 1, shows that the normal action of intra-abdominal pressure is to push the movable portion of the pelvic floor against the fixed portion, and so long as the relative positions of the two are maintained, and intra-abdominal pressure is balanced by the resistance of the fixed portion of the pelvic floor, no displacement occurs, in spite of the existence of the vaginal slit and the ring of loose tissue surrounding the movable position. The balance of forces is, however, liable to disturbance, with varied pathological conditions as results. Parturition is the chief source of injury to the pelvic floor. It stretches and renders still more lax the above-mentioned ring of loose tissue; it enlarges and softens the vaginal walls; it frequently causes tearing of the lower margin of the fixed portion of the pelvic floor, namely, the perineum; and it occasionally produces lacerations of the levatores ani, and other structures in the sacral segment. For Hart's original

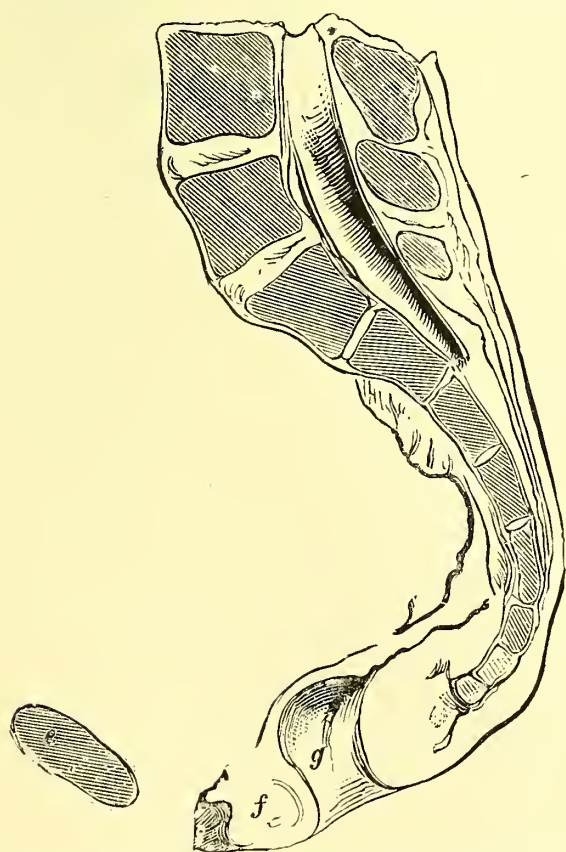


FIG. 2.—The sacral or supporting segment of the pelvic floor (Hart). *g*, Anus; *f*, perineum; *e*, symphysis pubis.

description of the structural anatomy of the female pelvic floor, upon which most subsequent descriptions are based, the reader is referred to Hart's *Structural*



*Anatomy of the Female Pelvic Floor* (Edin. 1881), and to Hart and Barbour *Manual of Gynæcology*.

It is convenient, in considering the lesions of the pelvic floor, to begin with those in which the fixed segment is involved, namely: 1. Undue descent of the whole pelvic floor; 2. Lacerations of the perineum; and 3. Rectocele.

The conditions in which both the movable and the fixed portions are at fault will next be discussed, namely, sacro-pubic hernia, or prolapsus uteri and cystocele. Finally, those lesions of the movable portion known as anterior and posterior vaginal enterocele must be described.

#### LESIONS IN WHICH THE FIXED PORTION IS INVOLVED

1. *Undue Descent of the Pelvic Floor*.—By the “pelvic floor projection” we understand the distance which the structures in the perineal region bulge or extend beyond a straight line drawn between the tip of the coccyx and the lower margin of the pubic symphysis, or, in other words, beyond the plane of the outlet of the bony pelvis. In normal women the average projection of the pelvic floor has been estimated by Foster at 2.5 cm., the measurement being taken with the patient lying down. It is increased in the erect and diminished in the genu-pectoral posture. The projection is increased during pregnancy, and is greater in parous than in nulliparous women, as is to be expected when we consider the amount of stretching and depression to which the sacral segment is exposed during parturition. In the absence of a special instrument, the bulge of the pelvic floor can be estimated clinically by measuring with a tape measure the distance from the top of the coccyx to the lower margin of the pubic symphysis. This averages about four inches, or, on straining, four inches and a half. Some parous women, who complain of pain in the back and hips, with dragging sensations in the pelvis, and other forms of discomfort, are found on examination to present no physical signs beyond an undue bulging or descent of the perineal structures. Upon measurement the distance from the tip of the coccyx to the symphysis may be found to be six inches, or even more. This incompetence of the pelvic floor is due in part to a general want of tone in the muscular structures, in which atrophic changes can sometimes be detected. Injuries to the levatores ani received during parturition are held by some to be of prime importance in the causation of the condition. Attempts at operative treatment are hardly justified in the present state of our knowledge. A perineal pad, supported by a belt fitting the hips like a hernia truss, generally relieves the symptoms. General treatment should be directed towards improving the muscular tone by special exercises, by hygiene, and by internal medication.

2. *Perineal Tears*.—For a description of the nature and causation of injuries to the perineum during labour, and of the measures demanded for their immediate repair, the reader is referred to the article, “Labour, Injuries,” in vol. vi. If lacerations of the perineum are not repaired by stitching within a few hours of their infliction, the torn surfaces do not become reunited, but are gradually covered with epithelium by a process of extension from their margins. There is some subsequent contraction of the cicatricial surfaces; this, however, does not by any means restore the lumen of the vaginal orifice to its original size. It is necessary to consider both complete tears of the perineum—those which divide the sphincter ani, and those which leave that muscle intact while more or less dividing the other structures which form the perineal body.

The anatomical result of a complete tear of the perineum is to unite the lower parts of the vagina and the rectum into a single tube, which corresponds with the cloaca of some lower animals. This traumatic “cloaca” is lined by rectal mucosa posteriorly, and by vaginal mucosa anteriorly, while at each side a triangular area,



with its apex pointing upward, is lined by the scar tissue covering the raw surfaces produced by the tear. Uniting the apices of these scar areas, the lower margin of the untorn portion of the recto-vaginal septum extends across the cavity, a narrow bridge of scar tissue dividing the rectal from the vaginal mucosa. The contraction of the sphincter and draws apart its torn ends, so that the position of these is marked by a depression on each side within the triangular area of scar tissue.

The most important physiological result of such a tear is inability on the part of the patient to retain fluid feces. Whenever the motions are unformed they escape, in spite of all voluntary efforts at retention, through the cloacal aperture. This causes the patient to favour a habit of constipation, which impairs her general health and makes her life miserable.

Other results of perineal tears are due to the impairment of the sacral segment of the pelvic floor as a supporting structure for the pubic segment. Though most marked in complete tears these results are shared by injuries, which, though they do not involve the sphincter, yet divide more or less completely the muscular and connective tissues forming the perineal body, or lower margin of the sacral segment. When combined with other causes of prolapsus uteri, cystocele, and rectocele, perineal tears assist in the production of these conditions. They are present in most cases in which these lesions exist, but they are also to be found in many women who have no other sign or symptom of defect in the pelvic floor, and alone they are quite insufficient to produce such. All tears which involve more than skin and mucous membrane should, however, be repaired. For though in young women there may be no appearance of secondary results, yet as life advances, and other factors come into play, serious changes may occur, which a simple and timely perineal repair would have prevented completely.

Operations for the repair of perineal tears, which have not been sutured immediately after labour, should be deferred until the torn surfaces are completely healed and the cicatrices have become firm and free from excessive vascularity. This, as a rule, requires from two to three months. Time should also be allowed in order for the patient to be up and about long enough to secure a good state of general health. This is necessary in order that healing of the operation wound may be favoured, and that the patient may well bear confinement to bed during the period of union. The operation should be done soon after the end of a menstrual period (if menstruation has returned), in order to allow as much time as possible for healing before the menstrual discharge reappears. The patient should be kept on light plain food for a week before operation, and should take aperients in sufficient quantity to produce at least one loose stool daily. A small dose of cascara sagrada (say  $\text{m x.}$ ) thrice daily with a mild saline on waking each morning is generally efficacious. An enema should be given two or three hours before operation, and should be repeated until the rectum is perfectly empty and clean. If there is any leucorrhœal discharge vaginal douching with very mild astringents and antiseptics should be practised for a few days previously.

The patient, when anæsthetised, should be placed upon a table in the lithotomy posture, a good light being secured. The legs should be supported by a Clover's crutch unless ample assistance is available. The object of the operation is to restore union between those structures which were in continuity before the laceration occurred, but which have become covered with a layer of cicatricial epidermis. If the tear is incomplete and the anus with its sphincter is intact, only one orifice, the vaginal, has to be restored to its ordinary dimensions. But it is necessary also to reconstruct the triangular mass of tissue known as the perineal body, and to do this it is necessary to unite between the vagina and the rectum lateral areas of tissue about  $1\frac{1}{2}$  inches wide at the skin surface, triangular in shape, their apices extending fully  $1\frac{1}{2}$  inches upward in the recto-vaginal septum. If the tear extends into the rectum it is further necessary to restore the integrity of the anal orifice, and above all, to secure accurate coaptation and sound



union between the divided ends of the sphincter ani. There are numerous ways of attaining the above objects, and it should be mentioned that it used to be usual and is still preferred by some operators, to raw the surfaces to be united by paring off and cutting away the scar tissue. We describe, however, only one way of dealing with perineal tears, a type of those operations in which no tissue is removed. The operations for incomplete and for complete lacerations demand separate description.

In operating for incomplete tears a horse-shoe or letter U-shaped incision is made round the hinder end of the vaginal orifice following the line

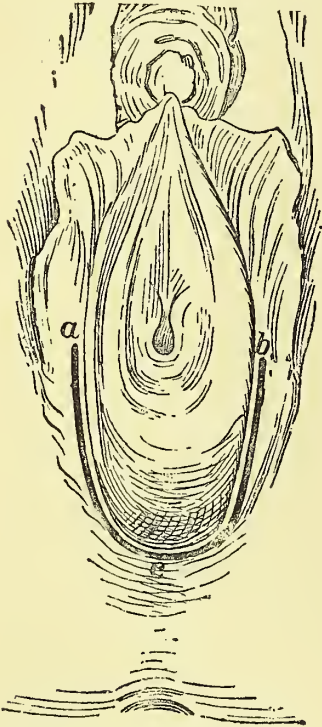


FIG. 3.—Incision for repair of incomplete perineal tear is commenced at *c* and carried to *a* and *b*.

where the skin joins the scar tissue (Fig. 3). The ends of the incision run forward a little farther than the scar tissue extends, and it must be remembered that these anterior ends of the incision mark what will be the posterior margin of the vaginal orifice when sutured. In a young woman likely to bear more children the incision must not be carried so far forward as is permissible in a woman more advanced in life, in whom the vagina may be almost closed with impunity, and in whom as a rule there is more need to form a strong perineal body. The incision is deepened by snips with the scissors which free the scar tissue and adjacent vaginal mucosa from the subjacent tissues. The flap of tissue raised and turned forward should be even in thickness, and great care should be taken not to perforate it with the scissors. The wound will ultimately be from an inch to an inch and a half deep in the centre (according to the extent of the tear), decreasing in depth at the sides as it nears the ends of the incision. Bleeding is checked by pressure, or by a stream of water at 118° F. allowed

to run over the wound from a douche can. If necessary bleeding points may be tied with catgut, as it is essential to prevent the formation of blood-clots within the wound after it has been closed. When bleeding has been checked the freed layer of superficial tissue is turned forward into the vagina, so that the wound, instead of being U-shaped, assumes the aspect of a letter V with another inverted over it  $\nabla$ . The sides of the V united in the middle line form the new perineal body. The sides of the inverted  $\Lambda$  brought together in the middle line merely add to the bulk of the new perineum. As above mentioned many operators cut away this tissue.

It is usual to close the wound with a series of interrupted sutures of silkworm gut. These are introduced with a well-curved needle so as to take a firm grip of the lateral aspects of the wound, and they are carried well to its bottom so as to prevent, as far as possible, the formation of pockets where blood-clot may accumulate. Such stitches are removed on the tenth or twelfth day, and answer their purpose fairly well. Many operators prefer to use a continuous suture of fine catgut hardened with chromic acid so as to resist absorption for about three weeks. The deeper portions of the wound are closed by stitches which are buried as the more superficial ones are inserted. The whole of the catgut is ultimately absorbed, and no removal of sutures is needed. This method completely avoids the puckering or pocket formation which is inevitable when it is attempted to unite two flat surfaces by a single row of sutures. It is



carried out as follows:—The middle of the edge of the flap to be turned forward into the vagina is seized with a pair of forceps and pulled upward and forward. A piece of “chromic acid” catgut about a yard long is passed by means of a fully curved needle (Nagedorn’s or Martin’s, with the appropriate needle holder) into the flap of tissue just below the tip of the forceps. It is tied in this position, and the loose end, being left about 6 inches long, is now used for holding up the flap instead of the forceps. The deep layer of sutures is now inserted from before backwards, the bottom of the wound being closed until its hinder end is reached, and care being taken that each stitch takes a firm grip of the tissues, but does not penetrate either rectum or vagina. The next layer of sutures is inserted from behind forwards, the first layer being buried and the wound rendered quite narrow and shallow. When the anterior or vaginal limit of the wound is reached the operator again turns backward and inserts the third or superficial layer of stitches, which finally closes the wound from before backwards. Some operators do not pass these stitches through the skin, but bring them out just under it; the majority, however, include the skin and draw its edges neatly together, so that the closed incision forms a single line from the vagina towards the anus. No dressing is necessary, indeed, the wound keeps dryer and cleaner if none is used. The knees should be tied together for a few days. The catheter may be needed for a day or two, though this is by no means the rule. It is said that if the skin be not stitched retention of urine is less likely to occur than otherwise. The bowels should be moved by aperient, enema, or both, on the third day, and regularly every day thereafter. The wound must be kept scrupulously clean, but vaginal douching is not desirable unless there is leucorrhœal discharge. (This should not be the case, as leucorrhœa should be cured before the operation is attempted). Stitches which cannot be absorbed by the tissues should be removed on the tenth or twelfth day. The patient may sit up at the end of two weeks, but should not rise till the end of three weeks unless the operation is very slight.

*Operation for complete Rupture.*—The operation for the repair of tears extending into the rectum differs somewhat from that above described. The incision is H-shaped instead of U-shaped. The transverse part of the incision (the cross-line of the H) runs along the edge of the recto-vaginal septum until the skin is reached on either side. The incision is then carried forward on each side of the vagina just as in operating for incomplete tear. Then it is carried backward on each side of the anus (so completing the upright lines of the H), so as to fully expose the torn and retracted ends of the sphincter ani. The recto-vaginal septum must be split for some distance, say three-quarters of an inch, so as to separate the vaginal and rectal walls well above the level to which they were torn. The forward and backward portions of the incision must be deepened accordingly by snips with the scissors until good flaps of tissue, freed from the sides of the “cloaca,” can be turned forward and backward respectively.

In suturing the integrity of the anal canal is first restored. This is best

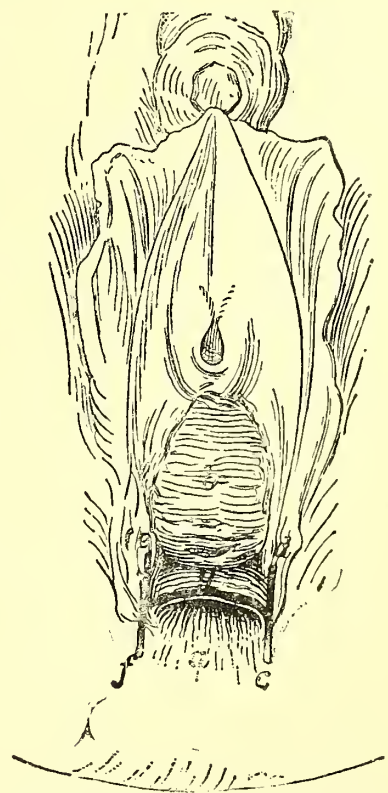


FIG. 4.—Incision for repair of complete perineal tear. *a*, anal wall; *b*, anterior vaginal wall; *c*, torn septum. Incision runs right and left forwards to *e* and *d*, and backwards to *f* and *c*. On suturing *f* and *e* come together, also *e* and *d*. (Hart and Barbour.)



done by interrupted sutures of catgut. The first suture is passed at the point of the tear which is highest up in the rectum, being inserted from the rectum into the wound on one side and from the wound back into the rectum on the other side, so that when the suture is tightened and tied the knot lies within the rectum and not in the wound. It comes away per anum when the portion of catgut within the tissues is absorbed. The next stitch is passed in the same way nearer the surface, and so on until the anal canal is once more complete, its front half being formed by the flaps of tissue separated from the sides of the cloaca by the backward portions of the incision. A finger introduced into the new anus enters it quite easily, for as yet the ends of the sphincter have not been firmly drawn together. This should next be done, and for this purpose it is well to use one or two stitches of silkworm gut passed so as to take a solid grip of the tissues at the hinder ends of the backward limbs of the incision. These should be tightened until the tip of the little finger is firmly gripped on gently passing it into the anus. The sphincter has shortened while thrown out of use; it lengthens again after its function is restored, and if the anus is not made apparently too tight at the operation, it will be found to be too loose after healing has occurred. The wound is now the same in shape and nature as that made in operating for incomplete tear, and may be closed either by interrupted sutures or by a continuous suture of chromic acid catgut as above described. In addition to the after-treatment necessary when the tear is incomplete, the operation for complete rupture must be followed by special attention to the rectum. It is no longer customary to keep the bowel at rest for several days. To give the best chance of perfect healing the rectum should be washed out every day for a week after the operation with a pint or more of warm water containing a little lysol, creolin, or boracic acid. The enema syringe should not be used for this purpose; but the fluid should be gently run into the rectum and out of it again through a yard or so of rubber tubing of the diameter of the little finger. A glass funnel is attached to one end of the tube, and the other end is gently passed into the anus, no nozzle being necessary. This washing of the rectum, combined with a mild aperient on the second evening after the operation, usually brings away a considerable amount of fæcal matter without any strain to the newly repaired anal canal and sphincter. If the patient be kept on fluid food for three or four days, and if the bowel has been well emptied before the operation, all will be well. If, however, from neglect of these precautions there is solid matter in the bowel, it must be carefully removed by enemata as occasion arises, a few ounces of olive oil being injected before the patient is allowed to expel a solid motion. The controlling sutures of silkworm gut should be removed about the twelfth day. The patient must be kept at rest for three weeks. Aperients must be used until the sphincter is completely and firmly united.

3. *Rectocele*.—While the anterior rectal wall forms a part of the fixed portion of the pelvic floor, the posterior vaginal wall loosely attached to it is a part of the movable portion of the pelvic floor. In displacements of the movable portion the vaginal wall usually is separated from the rectal wall, the latter remaining in or near its usual position. Sometimes, however, the rectal wall becomes pouched downward and forward, and, covered by the posterior vaginal wall, protrudes at the vulva as a swelling varying in size according to the presence in it or absence from it of fæces. Tears of the perineum, combined with the pressure of fæcal accumulations due to a constipated habit, are the factors that produce this condition, which is easily distinguished from prolapsus uteri by passing a finger into the rectum, when



it is recognised that a pouch of the latter organ occupies the swelling observed at the vulva. In uncomplicated rectocele the uterus, bladder, and anterior vaginal wall occupy almost their normal position. It must be remembered that prolapsus uteri can occur without rectocele, and as a rule does so; also that rectocele can occur without prolapse, and that the two conditions may exist together. The use of a ring pessary with a diaphragm, or a Hodge pessary with transverse bars, together with regular evacuations of the rectum, will relieve the condition as a rule. If the perineum is so torn that a pessary will not stay in position, the tear may be repaired by operation, and a pessary may then be worn. But if operative treatment is required the cure should be radical, and the pessary should be unnecessary. In some cases it is sufficient to tunnel well up between the rectal and vaginal walls when operating as above described (see "Perineal Tears"), and by stitching to throw the vaginal wall into one or two deep folds running upward from the perineal body. In other cases posterior colporrhaphy should be performed, a considerable portion of the posterior vaginal wall being removed so as to both narrow and shorten it. This is followed by perineorrhaphy. Other cases are best treated by raising a triangular surface on each side of the rectocele and closing these by folding each on itself ("Emmett's Operation"). The bases of these triangles are continuous with the ends of the wound by which the perineum is subsequently repaired.

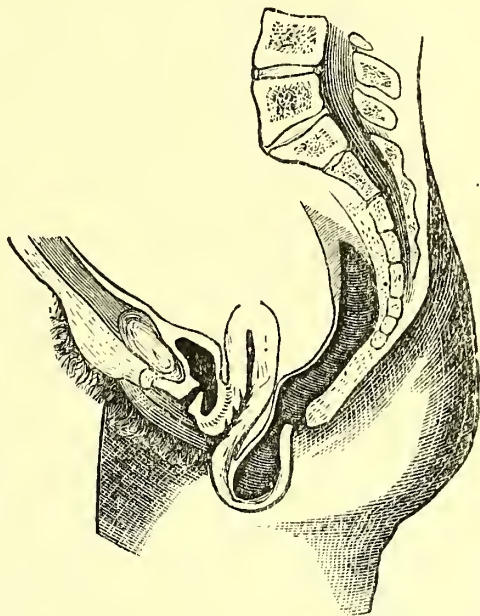


FIG. 5.—Rectocele. (Hart and Barbour.)

#### LESIONS IN WHICH BOTH FIXED AND MOVABLE POSTURES OF THE PELVIC FLOOR ARE INVOLVED.

1. *Prolapsus Uteri or Sacro-Pubic Hernia*.—"Prolapse of the uterus" is "not a mere uterine descent, but a downward displacement of the abdominal and pelvic viscera, along with the entire displaceable portion of the pelvic floor." It is thus a form of hernia (see Fig. 3). The movable portion of the pelvic floor, with the uterus and appendages, slides down upon the fixed portion, till a sac, lined by peritoneum and containing small intestine, is formed.

Three sets of causes combine to produce this condition—

1. Laxity and want of tone in the movable portion of the pelvic floor and in the ring of loose connective tissue which unites it with the fixed portion.

2. Impairment of function of the fixed portion as a supporting structure due to injury or to general laxity.

3. All circumstances which increase the intra-abdominal pressure which has to be resisted by the pelvic floor.

All circumstances which fall under headings 1 and 2 weaken the resistance of the pelvic floor. Those falling under heading 3 increase the displacing force to be resisted, and these latter are therefore the positive or prime causes of prolapse.

In a few cases congenital weakness of certain pelvic structures must be recognised as a predisposing cause, as instances occur in which young



women who have never been pregnant gradually or suddenly develop prolapsus after lifting weights or making unusual muscular efforts. Injuries received during parturition are, however, by far the commonest causes of lessened resistance on the part of the pelvic floor. Increased

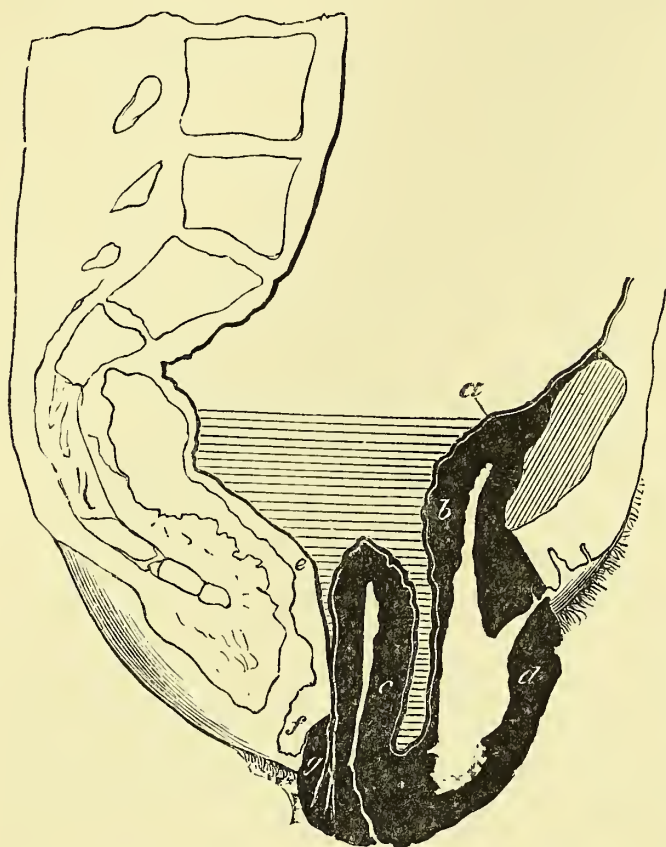


FIG. 6.—Prolapsus uteri. The displaced “movable portion” is shaded black. *a*, Peritoneum; *b*, bladder wall; *c*, uterus; *d*, anterior vaginal wall; *e*, rectal wall; *f*, perineal body (torn); *g*, posterior vaginal wall. The horizontal lines show the sac which is occupied by intestine. (Hart and Barbour.)

intra-abdominal pressure is occasionally due to the presence of abdominal tumours and other pathological conditions. In general, however, it is lifting heavy objects which increases the strain put upon the pelvic structures weakened by parturition. The abuse of the corset is an important cause of increased intra-abdominal pressure—more especially the early return to its use after parturition. Two or three weeks after labour, when all the tissues are soft, when muscular tone is low, when the uterus is large, the vaginal walls are lax, and the perineum is very frequently torn, the patient puts on her corsets, reducing her waist to the same size which it occupied before pregnancy began. The result is a raising of intra-abdominal pressure which is often enough under the circumstances to cause prolapse. Another fruitful cause of high intra-abdominal

pressure at a specially unsuitable time is the prolonged use of the obstetric binder. This appliance is excusable and comfortable for the two or three days immediately following labour, but after these have passed it is a useless and if tight a dangerous encumbrance.

Sufferers from prolapse complain of “down-bearing,” pain in the back, and pelvic discomfort, and also of the presence of a lump or swelling at the vaginal orifice. The soreness and discharge caused by excoriations upon the protruding mass is often the main trouble. There is also difficulty in passing water. In early cases the anterior vaginal wall is found protruding from the vulva, the cervix is near the vaginal outlet, the uterus is retroverted and large. The term “prolapse” is by some used for cases in this stage, the term “procidentia” being reserved for more advanced cases. In these the whole uterus enclosed in the inverted vaginal walls is outside the vulva and hangs between the thighs. The uterus is large and congested, the cervix gaping and hypertrophied, and the vaginal walls are often widely ulcerated. If the displaced organs be gently replaced in their normal position, and the patient, lying on her side, be then requested to strain down, the mechanism of prolapsus may be observed, the organs passing through, in a few moments, the phases which they have successively gone through as the condition developed, occupying, it may be, months or years in the process. The anterior vaginal wall first appears at the vulva. A finger placed in the vagina gives the information that as this occurs the uterus descends and at the same time assumes a retroverted position, its



long axis changing during descent and following the axis of the pelvic canal. The cervix next appears at and emerges from the vulva, the posterior vaginal wall finally descending as the whole uterus leaves the pelvis. The urethra, with the lower and hinder part of the bladder, and also the ureters, accompany the anterior vaginal wall in its descent (see Cystocele). The rectum, however, is separated, as a rule, from the posterior vaginal wall, and remains approximately in its usual position (see Rectocele).

True sacro-pubic hernia must be distinguished from hypertrophy of the cervix by examination with the sound. In the latter condition, though the cervix may appear at the vulva, the anterior vaginal wall and the bladder remain in their normal positions.

Cystocele may resemble prolapse, but in true cystocele the uterus retains its position, the lower and hinder portion of the bladder protruding at the vulva covered by the anterior vaginal wall.

In rectocele a finger passed into the anus runs into the pouch formed by the anterior rectal and posterior vaginal walls, the uterus, bladder, and anterior vaginal wall retaining their position.

The palliative treatment of prolapsus uteri consists, for the most part, in the use of pessaries. In many slight cases the use of a flexible rubber ring pessary, or of a vulcanite instrument of chosen size and shape, will give relief. In other cases a rubber bag fitted with a stopcock and inflated after insertion will render the patient comfortable. In more serious cases, before using a pessary, excoriations have to be treated with lotions and unguents, uterine congestion has to be reduced by astringent hot douches and rest in bed, and the uterus may have to be supported temporarily by packing the vagina with gauze. In certain cases repair of the perineum may be undertaken simply in order to enable the patient to wear a pessary, and not as an attempt at operative cure of the prolapse. In women in advanced life, and when for other reasons operative treatment is not desirable, the old-fashioned cup and stem pessary, supported by rubber tubing attached to an abdominal belt, may be very useful. As an alternative, when the pessary produces excoriations the vagina may be packed with tow or gauze every few days, a perineal pad and T-bandage being worn in order to keep the packing in position.

The choice of a line of operative treatment demands careful consideration in each case. It must be remembered that the following conditions are generally present:—

1. Enlarged congested uterus.
2. Relaxed ring of loose tissue between the movable and the fixed portions of the pelvic floor, involving separation of the rectum from the vagina, and separation of the uterus and bladder from the surfaces generally in relation with them.
3. Torn perineum.
4. Stretched and weakened "fixed portion," with probable lacerations of the levator ani.
5. Relaxed and enlarged vaginal walls.
6. Increased intra-abdominal pressure.

No. 1 can be improved by reposition, amputation of cervix, curetting, and other local treatment. No. 3 can be repaired. No. 5 can be improved by anterior and posterior colporrhaphy. But No. 2, No. 4, and No. 6 cannot be altered by operative interference.

Doubtless curetting, amputation of the cervix, narrowing the vagina by colporrhaphy, and repairing the perineum, will practically cure in many slight cases, but it is clear that these measures leave unaltered the more serious



factors in the causation of prolapse. We can go no farther, however, in the correcting of structural alterations by operative interference, and the failure of the above-mentioned measures in all but slight cases has led operators to go beyond the pathological indications, and to try to support the movable portion of the pelvic floor by any available means.

Thus we have to consider a set of operations which do not attempt to correct the causes of prolapse, but simply to substitute other means of support for the natural ones. We may mention *episioperineorrhaphy*, in which the edges of the labia majora are united, and *elytrorrhaphy*, in which sutures more or less completely unite the vaginal walls with one another so as to prevent their reversion. These measures attempt to support the uterus from below. Ventro-fixation, or the fastening of the uterus to the anterior abdominal wall above the bladder, attempts to solve the problem by suspending the movable portion of the pelvic floor from above. Two sets of operations designed for the cure of retro-deviations of the uterus are also employed along with other measures in operating for prolapse. These are vaginal-fixation, in which the anterior surface of the uterus is fixed to the anterior vaginal wall, the bladder being pushed up after a preliminary colporrhaphy; and those operations of which Alexander's is the type, in which the round ligaments of the uterus are shortened either by extra- or intra-peritoneal methods.

That none of these measures is entirely satisfactory is shown by the fact that some operators have performed vaginal hysterectomy, removing a considerable portion of the vaginal walls, together with the uterus, as a way out of the difficulty. This procedure, of course, leaves open the canal of the hernia, which has to be closed as far as possible by uniting the broad ligaments and the remainder of the vaginal walls. The operation is sometimes justified in patients past the menopause if completed by a perineal operation almost closing the vaginal outlet. It is, of course, most suitable if fibro-miomata are present in the uterus.

It is impossible to indicate a course of action suitable for every case. It is, however, clear that the lesions present should be treated in the first instance. Thus curetting, amputation of the cervix, and anterior colporrhaphy (see Cystocele) are almost always indicated. Perineorrhaphy is generally advisable (see Perineal Tears), and the removal of a portion of the posterior vaginal wall is also frequently desirable. The operations designed for the cure of retroversion are of doubtful value in prolapse. Some operators do Alexander's operation in addition to the measures just mentioned, while others, when removing part of the anterior vaginal wall, take the opportunity of pushing up the bladder and including part of the anterior wall of the uterus in the stitches which close the vaginal wound. The fundus of the uterus should never be fastened into this position for fear of the consequences should pregnancy occur subsequently. It will probably be found that vaginal fixation and shortening of the round ligaments are of less value in prolapsus than ventro-fixation. This measure, though weak theoretically, is of practical utility, and is free from evil results should pregnancy follow, provided only that care is taken to leave the fundus free to rise. The anterior wall below the fundus is the portion of the uterus that should be sutured to the edges of the wound in the abdominal wall. The operation can be done conveniently after plastic operations on the vagina and perineum have failed, or it may be performed in bad cases as soon as uterine congestion and vaginal ulcerations have been relieved by rest in bed and local treatment.

2. *Cystocele*.—When the anterior vaginal wall descends with the bladder



and urethra in the first stage of uterine prolapse the tumour which appears at the vulvar outlet may doubtless be termed a cystocele. This, however, is not the strict sense in which the term is accepted. For true cystocele, namely, a protrusion at the vulva of the lower and hinder portion of the bladder covered by the anterior vaginal wall, has a clinical existence apart from uterine prolapse or descent of the whole movable portion of the pelvic floor. In short, prolapse cannot occur without cystocele of a kind, but true cystocele can occur without prolapse. The cause of the condition is undue laxity in the connections between the bladder and the uterus and adjoining sutures, combined with failure in its supporting function of the lower margin of the sacral segment, due to tearing of the perineum. The diagnosis is made by bimanual examination and the use of the sound in the bladder. The uterus and fundus of the bladder occupy their usual position, the lower portion of the bladder being pouched downward and backward. The main,

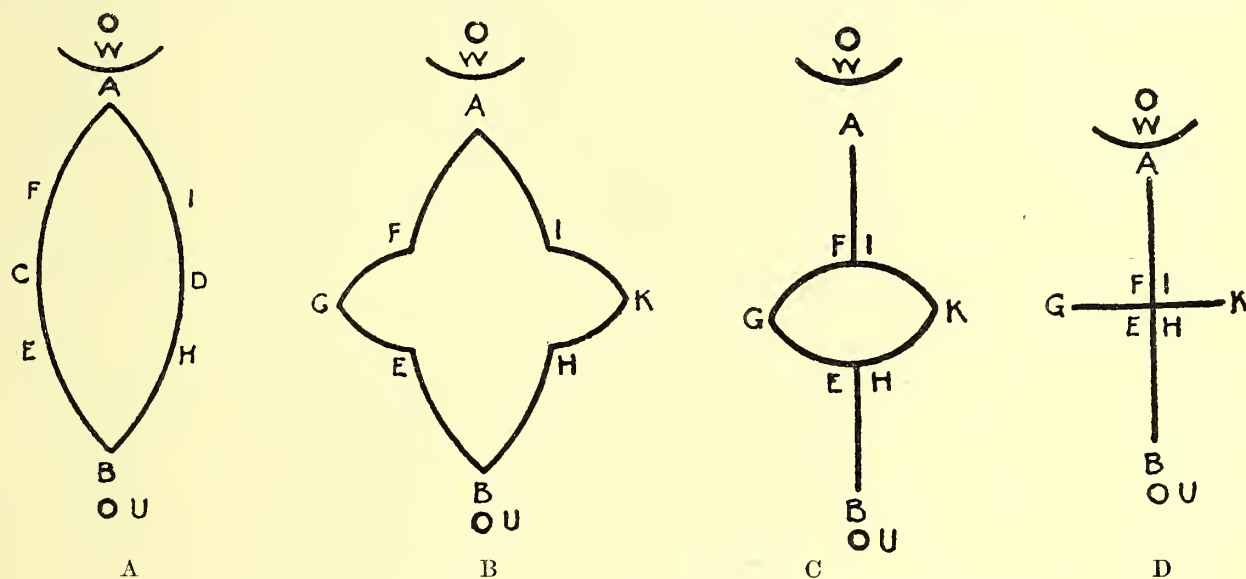


FIG. 7.—Anterior colporrhaphy. w, Urethra; ou, os uteri. (Skene Keith.)

symptom is difficulty in completely emptying the bladder, with cystitis as a secondary result, especially common in the senile form. A ring pessary with diaphragm, or a Hodge pessary with transverse bars, may relieve the patient of all difficulty. A perineorrhaphy may be necessary in order to make it possible to wear the instrument. If, however, any operative treatment is adopted, it is advisable to perform an anterior colporrhaphy which will render an instrument needless. There are many ways of doing the operation. It must be remembered, whatever the method chosen, that the anterior vaginal wall is both longer and wider than it should be, and that it must, therefore, be shortened as well as narrowed. A useful method is that of Currier. An area of mucous membrane is rawed, as shown in Fig. 7 A, broad enough to cause when C and D are brought together sufficient narrowing of the vaginal wall. In order to give the necessary shortening further areas are denuded, as shown in Fig. 7 B. When all bleeding has been stopped by means of pressure and hot water, with ligatures if necessary, the round surfaces are united, as shown in Figs. 7 C and 7 D. Interrupted suture may be employed, or two continuous sutures of chromic acid catgut may be used. Suture 1 runs from A to F, and is then left long. Suture 2 runs from G to F, and is then left long. The portion of No. 1 is now used again, and runs from E to B, where it is finally tied off. The remainder of No. 2 now closes the wound by running from I to K, and the operation is complete. Coughing, vomiting, and straining must be avoided while union is taking place. No dressing is necessary, nor is vaginal douching desirable, but rest in bed should be continued for quite two weeks.



FAULTS IN THE MOVABLE PORTION OF THE PELVIC FLOOR

*Vaginal Enterocele.*—This is a very rare condition which, strictly speaking, is a form of hernia, since its essential feature is the formation of a sac lined by peritoneum and occupied by small intestine or omentum (or occasionally by ovary), which protrudes through an opening in the pelvic floor into the vagina. The condition is perfectly clearly distinguished from prolapsus uteri by the fact that in vaginal enterocele the hernia descends through the movable portion of the pelvic floor, and is not a descent of that portion of the floor *en masse*. There are two varieties of vaginal enterocele, the anterior and the posterior.

In the posterior variety coils of intestine separate the rectum from the posterior vaginal wall, the pouch of Douglas being deepened and extended. Sometimes the hernia penetrates the muscular wall of the vagina, and its peritoneal sac is covered only by the vaginal mucosa. The swelling can be recognised, both by vaginal and by rectal examination, as a mass of intestine descending between the fixed and the movable portions of the pelvic floor.

In the anterior variety the sac of the hernia is formed by extension of the utero-vesical pouch intestine descending between the uterus and the bladder. The uterus is displaced backwards, and on vaginal examination the sac with its contents is felt bulging into the vagina in front of the cervix, having passed through the movable portion of the pelvic floor.

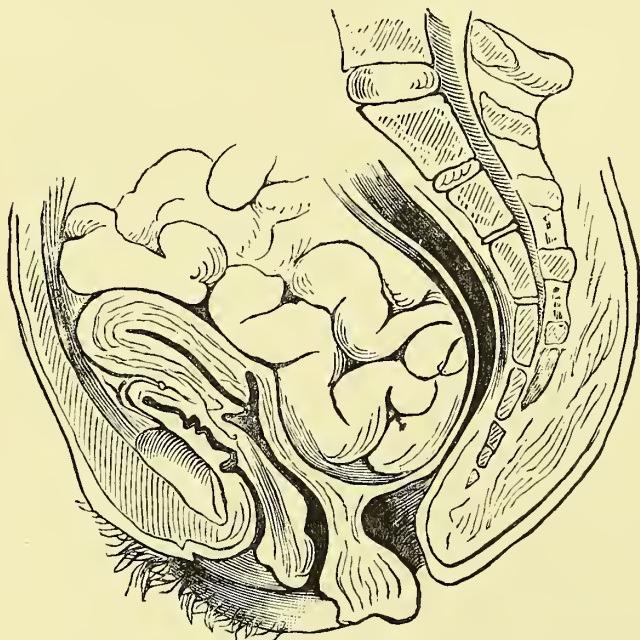


FIG. 8.—Posterior vaginal enterocele. (Hart and Barbour.)

The main factor in the causation of vaginal enterocele is increased intra-abdominal pressure. There is usually a history of a fall, of straining, or heavy lifting, or of parturition.

In slight cases it is generally possible to select a pessary which will prevent discomfort. In more pronounced cases of the anterior variety an anterior colpotomy will allow the operator to push up the sac without cutting the peritoneum. By suturing the anterior surface of the uterus to the upper portion of the anterior vaginal wall the canal is obliterated, and the future descent of the sac is rendered impossible. In posterior enterocele the appropriate operative treatment consists in rawing the upper portion of the posterior vaginal wall and folding it upon itself transversely, so as to shorten the wall and bring the cervix close to the reflexion of the peritoneum from the wall of the rectum. As an alternative the posterior fornix may be freely opened and the sac closed up by sutures.

**Pelvis—Diseases of the Cellular Tissue.**

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I. PELVIC CELLULITIS

As the name implies, this consists of an inflammation of the cellular tissue



of the pelvis. Until comparatively recently it was generally known by the name of parametritis, a term which now, fortunately, has almost entirely been given up as unscientific and confusing.

The cellular tissue of the pelvis has a well-defined distribution. It may be described as mainly forming a ring round the cervix at the utero-vaginal juncture (the parametrium); from this ring processes of tissue pass in several defined directions. Backwards to the sacrum, the utero-sacral ligaments, laterally between the layers of the broad ligaments, and forwards along the round ligaments. Each process is shut off by the so-called lines of cleavage described by König, a point of great clinical importance, as giving the direction of the inflammatory exudations when the cellular tissue is infected.

Below it is bounded by the pelvic fascia upon which it may be said to lie, while above it is loosely covered by the peritoneum. Ordinarily it measures about one-third of an inch in thickness, but during pregnancy is enormously increased.

Primary inflammation of the cellular tissue is always infective. Most commonly a direct inoculation of the parametric ring through lacerations of the cervix or upper end of the vagina, but rarely also through absorption—through the uterine wall, as shown by Polk and Lewers. It will thus be evident that the majority of cases occur after full time labours, where lacerations of the cervix are prone to occur, or after operative procedures on the uterus where the cervical continuity has been interfered with.

*Clinical Features.*—Acute pelvic cellulitis may be ushered in by a well-marked rigor, which in puerperal cases occurs about the second or third day after delivery. In a large number of cases, however, the onset is slower, a subacute inflammatory state is here primarily present, which is recognised only by an elevated evening temperature,  $100^{\circ}$  to  $101^{\circ}$  F., and a persistent increase in the pulse-rate, unassociated with any constitutional symptoms whatever. After many days the subacute may pass into the acute condition, a change which is marked by a rigor and constitutional symptoms. Pain is unusual in uncomplicated cases, and betokens involvement of the peritoneum. The symptoms vary materially according to whether there is suppuration; should pus form, the patient soon assumes an emaciated hectic appearance and looks extremely ill. The skin is dry, tongue furred, bowels constipated, and temper at first irritable, but later the disposition tends towards the phlegmatic. The pulse remains persistently over  $100^{\circ}$ , and the temperature swings violently between  $100^{\circ}$  and  $104^{\circ}$  F.

Where there is simple exudation without suppuration, the symptoms are milder; perhaps the most important difference is the temperature, which is rarely over  $102^{\circ}$  F. Should the exudation be present in the neighbourhood of the psoas and iliacus muscles the patient will usually be noticed to keep the thigh flexed on the abdomen to relieve tension on the muscle. Care should in these cases be taken to prevent permanent contraction of the knee-joint by daily straightening the knee by lifting the foot.

The course of the disease is as in other inflammations, viz. resolution and absorption or abscess formation with subsequent rupture and evacuation of the pus. Absorption is extremely slow, and thickened deposits may be found in the pelvis many months after all symptoms have subsided. Abscess formation occurs in a considerable number of cases. It usually points from the seventh to the twelfth week. The situation where pointing may occur must necessarily vary according to the line of inflammatory exudation which is influenced by the lines of cleavage of the cellular tissue. Most frequently the area of infiltration is between the



layers of the broad ligament, it then passes forward under the peritoneum to the anterior abdominal wall above Poupart's ligament, and then outwards and backwards into the iliac fossa.

Abscesses developing posteriorly fill the postero-lateral part of the pelvis, then pass along the psoas and iliacus, and then sink into the true pelvis. When the exudation passes anteriorly, pointing usually takes place on the anterior abdominal wall slightly above Poupart's ligament. When posterior, however, burrowing is very extensive, and may pass through the sciatic notch or be found in Scarpa's triangle, having found its way along the course of the femoral vessels. Extremely rarely is it that an abscess ruptures into any of the hollow viscera, nearly all such cases are perimetric in their origin.

*Diagnosis.*—This must depend on the recognition of the inflammatory exudation by the bimanual method of examination. In the majority of instances from the implication of the parametrium the intra-vaginal portion of the cervix will be found fixed, shortened, or obliterated, while passing from it will be felt a diffuse brawny mass depressing the lateral fornix vaginae. Should the exudation have spread anteriorly, the brawny swelling will be felt under the anterior abdominal wall above Poupart's ligament and extending into the iliac fossa, while in posterior exudations a diffuse fulness and hardness of the posterior pelvic segment will be felt on the affected side, and rectal examination will show an infiltration in close proximity to the gut, and in some instances surrounding it. The uterus may from the size of the exudation be pushed to one or other side, but usually retains a certain amount of mobility. Examination may or may not be painful, firm pressure on the exuded mass usually elicits tenderness, particularly should suppuration have commenced. Chronic exudations frequently are entirely painless on pressure. The differential diagnosis from perimetric exudations is sometimes extremely difficult and will be described under that affection. From other pelvic swellings, such as tumours and hæmatomata, the presence of constitutional symptoms of fever will distinguish in acute and subacute cases, while chronic inflammatory deposits can usually be told by the previous history of fever, etc.

The prognosis is usually favourable. When no suppuration occurs, the fever usually subsides in a few weeks, and complete absorption, though slow, eventually takes place. Pus formation is distinguished by the maintenance and increase of the fever, which is present until evacuation takes place. After the pus has escaped, convalescence almost immediately occurs, chronic sinuses being rarely met with. Extension of the inflammation to the peritoneum of a severe type rarely occurs.

*Treatment.*—Septic inflammation having occurred, it is doubtful if treatment can materially modify this course. Locally, hot vaginal douching, 110° to 115° F., is of value in assisting absorption should suppuration not have occurred, while the absorption of chronic inflammatory deposits is expedited by ichthyol in the form of vaginal plugs (10 per cent in glycerine), and inunction over the pelvic brim (15 per cent in lanoline or 5 per cent in vasogen).

Should suppuration occur, little can be done but wait till signs of pointing occur, then free incision and drainage for a few days will be curative. Usually pointing occurs above Poupart's ligament externally, but in rare cases it may be in the vaginal fornix, hip, or perineum. Incision should never be attempted till distinct evidence of fluctuation is present.

During the prolonged period of suppuration great attention must be



directed to the conservation of strength by good feeding. The internal administration of drugs is valueless, opium is specially to be decried unless great pain is present, as it only interferes with digestion and locks the bowels, regular evacuation of which is most important and must be carefully attended to.

## II. PELVIC PERITONITIS

Is the term used for localised inflammation of the peritoneum within the pelvis. It is also known as perimetritis, and is perhaps the most common pelvic inflammatory affection.

Recent investigations tend to show that it is probably never primary in its origin, but is the result of extension of other pelvic disease, and it may thus be frequently looked upon as indicative of pelvic mischief which has hitherto given no indication of its presence.

By far the commonest cause is inflammation of the mucosa of the Fallopian tubes. From its direct continuity, on the one hand, with the vagina and uterus, and the peritoneum on the other, extension of inflammation is prone to travel by this route, and thus infections of all types—septic, tubercular, or gonorrhœal—by this means reach the peritoneum. New growths of the ovary and inflammation of that organ are known sources of peritonitis, probably due to the fact that the ovary is not covered by peritoneum, but lies free in the peritoneal cavity. Simple uterine fibro-myomata, on the other hand, being extra-peritoneal, seldom gives rise to pelvic inflammation.

Simple injury without septic infection is probably never a cause.

Direct extension of inflammation of septic origin may occur through the wall of the uterus, but more frequently travels along the tubes. Thus operations on the body of the uterus, such as curettage, if carelessly undertaken, are a common source of pelvic peritonitis. Operations on the cervix more frequently give rise to cellulitis.

Extension of inflammation from pelvic cellulitis is common, as also from appendicitis. So-called chills at menstrual periods, and sudden arrests of that function, though commonly thought to result in pelvic inflammation, cannot be considered as probable causes.

*Pathology.*—From the exudations of plastic lymph adhesions form the most characteristic feature of the disease. Along with the lymph serum is also effused, and together they may form a thickened mass of exudation. As the serum becomes absorbed the lymph stiffens, and a hardened mass remains. This may gradually be absorbed, but frequently permanent adhesions result. In some instances pus instead of serum is formed, and localised peritoneal abscesses follow. From the tube being the common seat of original inflammation, the situation of the exudation, if small, is usually behind the uterus which, along with the tubes and ovaries, are matted together on one or both sides. Intra-peritoneal abscesses walled in by adherent organs tend to rupture into the hollow viscera, and form fistulæ which may last for many months or even years. Being due to diseased conditions of the organs, recurrent attacks of pelvic peritonitis are common, and indeed form a valuable diagnostic indication of the presence of tubal disease which otherwise shows no signs of its presence.

*Clinical Features.*—Acute pelvic pain associated with fever and frequently vomiting, and more or less intestinal distension, are the characteristic symptoms. The patient lies on her back with the legs drawn up and assumes an anxious expression. The temperature is usually high, 103° F., but there is seldom manifestations of a rigor. Micturition is frequently



painful if the inflammation has extended forwards to the covering of the bladder. Diarrhoea is very common, though constipation may be present.

After the subsidence of the acute attack, pain may only be experienced on certain movements, and the patient rapidly returns to apparent health, but frequently, as has been stated, recurrent attacks are prone to occur.

On examination during an acute attack there is extreme tenderness to touch over the lower abdomen, with great rigidity of the abdominal muscles. Bimanual examination is practically only possible under an anæsthetic, when the presence of the exudation, and fixation and matting will be made apparent. The retro-uterine pouch will be felt to be filled with exudation. The uterus may with difficulty be mapped out from the surrounding structures to which it is firmly fixed, or in some cases may be felt to be tilted above the symphysis pubis. Frequently, the entire vaginal roof appears as if it had been boarded over, while intra-vaginal cervix is free and movable. After the acute conditions have passed and the general exudation been absorbed, there is usually left an area of thickening and adhesion at the original site of the inflammation. This may closely resemble a cellutic exudation, and the differential diagnosis may prove extremely difficult. The history of the case, the presence of severe pain, and the free intra-vaginal cervix will, however, materially help in the differentiation.

*Prognosis.*—The prognosis must at all times be guarded and indefinite. Though recovery from the acute attack may usually be looked for, the source of the inflammation still remains, and future attacks liable to occur. Further permanent adhesions causing much pain and discomfort are liable to persist, while interference with intestinal movements may cause much trouble with the bowels. Complete recovery may take place, and this greatly depends on the origin of the inflammatory attack.

*Treatment.*—This may be divided into medical and surgical.

Medically, during an acute attack, our main endeavours must be directed towards the alleviation of pain by means of morphia and local application of hot fomentations. The bowels should be emptied by enemata and a brisk saline purge administered. The dieting and general treatment must be carried out as in general peritonitis, *q.v.*

Surgically, during an acute attack there is usually little to be done. Should there be evidence of fluid bulging into the vagina, this must be at once evacuated through the fornix, but beyond this surgical interference is seldom called for during the acute stage.

After the acute attack has subsided, the absorption of exudation and removal of adhesions must be assisted by hot vaginal douching and the application of ichthyol by means of vaginal plugs, 10 per cent in glycerine and 15 per cent in lanoline, for abdominal inunction.

If on vaginal examination any tenseness is discovered in the pouch of Douglas or the lateral fornices, a vaginal incision should be made to allow of the escape of pus. Drainage by gauze will be necessary for some time. The progress of an inflammation is frequently limited or arrested by such treatment. Furthermore, great relief from pain is bestowed on the patient, and the formation of fistula prevented. If the patient has suffered from repeated attacks, and on examination a swelling of such size is discovered as to render it probable that the patient's condition arises from an occluded and distended Fallopian tube, operation should be urged.

As to the procedure now to be employed opinions differ. Abdominal section, followed by the breaking down of adhesions, and the ligature and removal of the implicated tube, is the older and more generally adopted measure. In 1886 Pean first advocated removal of the uterus and the diseased appendages by the vaginal route. This method has now many advocates. Others leave the uterus untouched and content themselves with removal of the diseased tube or tubes per vaginam.



Against this it is urged, that as the disease in most cases extends to the tubes, and through or by them to the pelvic peritoneum from the uterine mucosa, if the latter is not removed, the original source of mischief remains to continue the patient's distress.

#### CYSTS AND TUMOURS OF THE BROAD LIGAMENT

*Malformation of the broad ligament* is a rare condition generally associated with a similar abnormality affecting the ovaries, Fallopian tubes, and uterus. The broad ligaments may also be entirely absent or rudimentary, or unequally developed on both sides.

*Cysts of the broad ligament* may arise from the hilum of the ovary, from which they spread between the layers of the broad ligament; or they may originate in the broad ligament independently of the ovary. These may be situated on the surface of the ligament, or may be deeply placed between its folds. The origin of the superficial cysts is unknown, those lying at a deeper plane spring in many cases from changes in the tubules of the parovarium. The former are small and frequently multilocular, the latter are thin walled, and, as a rule, unilocular. The contents of these cysts is a clear or opalescent fluid of a specific gravity 1002·6, albumin free, but holding chlorides in solution. They are lined by ciliated columnar or by cubical epithelium. Doran and Sutton have described a papillary cyst of the broad ligament identical with a similar tumour of the ovary. In rare cases cysts have arisen by distension of the hydatid of Morgagni; those are small and clinically unimportant. Hydatid or echinococcus cysts have been described as arising in the pelvic cellular tissue, and always in close relation to the intestines. The invasion of the parasite results in a chronic inflammation characterised by round elastic tumours which are slightly movable and not painful. A positive diagnosis can only be made by a microscopical examination of the contents of the cysts. If the tumour is situated low down in the pelvis it should be attacked by the vaginal route, the sac emptied, the lining membrane removed, and thin gauze introduced into the cavity. When the peritoneal cavity has to be opened into, Pozzi advises that the opening over the cyst should be packed with gauze from twenty-four to forty-eight hours. Adhesions form in this period, after which the cyst can be opened without danger of infecting the peritoneal cavity.

*Varicocele of the broad ligament* is most frequently met with on the left side, for the reason that the left ovarian vein has a long course to run before it joins the left renal vein. Furthermore, the left ovarian vein is larger than the right, as it is joined by two colico-spermatic veins from the descending colon. The varicose masses may form a tumour as large as a walnut. Clinically they manifest themselves by sensations of dragging and pain in the pelvis, which symptoms are immediately relieved by the patient assuming the recumbent posture. Thrombosis sometimes occurs, and phleboliths are not unfrequently found in these tumours. Abdominal section and removal of the masses between ligatures is the correct treatment.

*Actinomyces of the Broad Ligament.*—In a case described by Grainger Stewart and Muir, invasion by the organism seems to have occurred per vaginam; the Fallopian tubes, ovaries, and pelvic cellular tissue were secondarily involved.

*Lipomæ of the meso-salpinx* are of not rare occurrence. They are usually found near the under surface of the tube; they vary in size from a bean to a walnut, and are of no clinical importance.

*Fibromata and fibromyomata of the broad ligament* are occasionally found independent of the uterus or ovary. Non-striped muscle fibres occur in the broad and round ligaments, and from those such tumours arise. They occur most frequently on the right side, and rarely attain to a large size. Sometimes they are pedunculated, but more commonly are sessile.

*Sarcoma and carcinoma* of the broad ligaments are usually the result of extension of the disease from adjacent parts, but in some rare cases the lesions have been primarily in the broad ligaments. Diagnosis can only be made by microscopical examination.

#### Pelvis—Hæmatocele and Hæmatoma.

I. INTRA-PERITONEAL FORM OF HÆMATOCELE.—Of this two varieties are distinguished, differing only in the rapidity with which they are formed. If the hæmorrhage be abundant no definite tumour is formed, and as a rule



the patient rapidly succumbs. This form is known as a "non-encysted hæmatocele." If the hæmorrhage is comparatively small in quantity, or slow in accumulating, it gravitates towards the pouch of Douglas, where its presence produces a localised peritonitis, which results in the formation of adhesions, and subsequently of a cyst. The walls of such a cyst are formed anteriorly by the uterus, posteriorly by the rectum and the peritoneum lining the posterior wall of the pelvis, laterally by the utero-sacral ligaments. The roof is supplied by the coils of the intestines, which have become adherent to the fundus uteri, the broad and round ligaments, the ovaries and tubes, and the peritoneum lining the lateral and posterior walls of the pelvis.

II. EXTRA-PERITONEAL FORM OF HÆMATOCELE OR HÆMATOMA.—The hæmorrhage in such a condition is found in the cellular tissue which surrounds the uterus and the other pelvic organs. The commonest site is between the layers of the broad ligaments, as the blood-vessels in this situation are large, and, being surrounded by looser areolar tissue than elsewhere, are least supported. Sometimes the blood invades the cellular sheath which surrounds the rectum. Owing to the greater resistance to the escape of blood the tumours of this group are generally much smaller than those of the intra-peritoneal class, but in some cases the pressure of the effused blood is sufficient to strip the peritoneum from its usual attachments, and it may be sufficiently powerful to rupture this membrane, and a secondary hæmorrhage may occur into the peritoneal cavity.

*Causes* may be divided into two classes. First, hæmorrhage arising from rupture of one of the various forms of extra-uterine gestation. This is by far the commonest source of pelvic hæmatoceles, and some have gone so far as to regard it as being their only origin. Second, intra-abdominal hæmorrhage may arise from injuries to the viscera or rupture of an aneurysm of the abdominal aorta or coeliac axis. Such hæmorrhages naturally gravitate to the pelvis. Again, regurgitation of menstrual blood through the Fallopian tubes is regarded by many authorities as a possible cause. Rupture of a varicose vein in the pampiniform plexus is described as originating a hæmatoma by Bandl, Scanzoni, and others. Cullingworth describes a case where such a condition arose from rupture of a varicose vein inside the Fallopian tube. Dalbeau, Bernatz, and others hold that a hæmorrhagic type of pelvic peritonitis exists which is not a serious condition, as the bleeding is small in amount. Finally, some causes arise from morbid conditions of the blood, such as occur in purpura and following the exanthemata.

*Symptoms*.—The symptoms of a non-encysted hæmatocele are those of a ruptured extra-uterine gestation (see art. "ECTOPIC GESTATION"), and need no further notice here. Those of the encysted variety are to some extent the same, but less in severity—this of course being modified in accordance with the suddenness of onset and the amount of blood extravasated.

In cases where the effusion is considerable the symptoms approximate to those of the non-encysted type, followed by signs and symptoms of localised peritonitis. On the other hand, when the symptoms develop gradually, and where the effusion is small in amount, serious difficulty exists in coming to a correct diagnosis as to the condition. Some time must elapse before the blood becomes so consolidated as to be defined; further variations occur in firmness according to the duration of the tumour. Examination per rectum and per vaginam should be made, as thus in the intra-peritoneal form the best idea as to the size, consistence, and relations of the tumour can be obtained. When the effusion has occurred into the pouch of Douglas the uterus is pushed upwards and forwards, while the



posterior wall of the vagina is moved downwards, and in extreme cases may reach the vulva. In the extra-peritoneal form the tumour will be felt lateral in position and fixing the uterus, and if seen in the latter stages the diagnosis between this condition and pelvic cellulitis may be impossible. The history of onset is helpful sometimes.

*Diagnosis*, whether intra- or extra-peritoneal, is determined by the phenomena of internal hæmorrhage (see "ECTOPIC GESTATION"). Rupture into the peritoneal cavity of a pyosalpinx or pelvic abscess may give rise to very similar symptoms, but in such cases the resulting peritonitis is very much more intense and the pain more intolerable. The difficulty of a differential diagnosis between long-standing pelvic hæmatocele and pelvic cellulitis and peritonitis has already been alluded to.

Pelvic hæmatocele is much rarer than pelvic cellulitis, and the latter is usually a consequence of parturition. The former tumour is soft at first, gradually becoming hard, the latter hard from its inception. Febrile symptoms follow the hæmatocele, but precede the cellulitis. Tumours of the uterus and ovaries sometimes simulate hæmatoceles, but they are to be differentiated by the absence of urgent symptoms at the onset, slow growth, better definition, and generally by their mobility. Sudden hæmorrhage into the cavity of a large ovarian cyst may produce symptoms very akin to those of a hæmatocele, but in such a rare condition the existence of a tumour previous to the effusion could be ascertained.

Extra-peritoneal hæmorrhage is to be differentiated from intra-peritoneal hæmorrhage by the fact that the former lies to the side of the uterus, the latter posterior to that organ. The former has its boundaries more sharply defined, and the uterus is pushed to the opposite side. Excluding rupture of an extra-uterine gestation, the determination of the cause of the hæmorrhage is often difficult.

*Prognosis*.—When arising from rupture of an ectopic pregnancy the prognosis has already been considered (see art. "ECTOPIC PREGNANCY," vol. iii.). In cases arising from other causes prognosis on the whole is good, provided proper treatment has been adopted. Danger exists from the possibility of suppuration occurring in the blood cyst from chronic peritonitis, also from the extensive formation of adhesions, which in turn may act injuriously on the ovaries, tube, and bowels.

*Treatment*.—Non-encysted extravasations when arising from rupture of an ectopic pregnancy do not require consideration here. But as practically all intra-peritoneal effusions are at first non-encysted, the desirability of laparotomy must depend on a consideration of the condition of each individual case. Where operative procedures are not deemed necessary, the patient should be made to assume the recumbent posture. Pain and restlessness may be combated by full doses of morphia. The local application of an ice-bag over the hypogastrium is a valuable method of checking the hæmorrhage. Internally lead acetate, ergot, suprarenal extract (5 grains of the medullary portion of the gland) may be administered. The patient should be kept for some time on a low diet; all hot drinks should be avoided. Frequent examinations of the patient are undesirable, as in fact are all procedures which prevent the absolute repose demanded in the early non-operative treatment of pelvic hæmatocele. When febrile and other symptoms point to suppuration, evacuation of the pus should be effected, and if possible per vaginam. Free drainage should be obtained, care, however, being taken that existing adhesions are not broken down lest the peritoneal cavity becomes invaded. When the condition has become chronic the patient should avoid all severe strains or exertions lest the hæmorrhage should



return; complete rest should be insisted on during each menstruation. Attention to the bowels is necessary. For anæmia iron is indicated, and the administration of potas. iodidi helps in the absorption of the effusion; this may be assisted further by the application of blisters over the seat of the lesion.

### **Pemphigus.**

*DEFINITION.*—A disease characterised by a generalised eruption of vesicles and bullæ, usually arising from apparently healthy skin, and always containing clear serum at first.

It will in all probability be found when we begin to know something more of the etiology and pathology of the group of symptoms which we at present collect under the name of pemphigus, that we have hitherto included several distinct diseases under a single name.

Some advance was made by the separation by Tilbury Fox, and later by Duhring, of the disease now known as dermatitis herpetiformis, though the advantage here gained is not so great as it should be, owing to the fact that complete agreement does not exist as to the conception of this disease.

There remain after the separation of this disease and those eruptions which are merely phases of other well-defined diseases, such as pemphigus syphiliticus and leprosus, better called bullous syphilis and leprosy (see under these diseases), five fairly distinct varieties of pemphigus:—

- (1) Pemphigus acutus neonatorum.
- (2) Pemphigus acutus malignus.
- (3) Pemphigus chronicus vulgaris.
- (4) Pemphigus foliaceus.
- (5) Pemphigus vegetans.

In this article will also be described the disease previously known as congenital traumatic pemphigus, but now more usually and appropriately as epidermolysis bullosa hereditaria.

(1) PEMPHIGUS ACUTUS NEONATORUM.—One or two cases have been recorded in which the child has been born with the eruption already developed, but more usually it appears from the third to the fourteenth day. The eruption consists entirely of round or oval bullæ, which appear suddenly on previously normal skin, and are at first tense, crystal clear, and filled with a straw coloured fluid.

There is usually a very slight zone of hyperæmia around the blebs, but this does not become marked unless the case becomes infected with pyogenic organisms. The younger the child the more fragile are the bullæ, probably from the extreme delicacy of the horny layer. In very young children, therefore, all the bullæ occurring on parts which are subject to pressure or friction are rapidly broken, and large areas of excoriated skin result. The denuded surface after bursting of the bulla is red and weeping, but in uncomplicated cases it becomes rapidly covered by a new horny layer.

There seems to be no special site of predilection for the occurrence of the bullæ, and the disease usually runs its course without fever or any other disturbance of the general health in from one to eight weeks. Sometimes, however, the cases are severe, and become complicated by intestinal catarrh, marasmus, or lung disease.

*Etiology and Pathology.*—There seems to be little doubt that the disease is septic in its nature, but whether it is definitely contagious or not is a matter of uncertainty. In one case a woman suffering from a septic affection gave birth to a child which exhibited the eruption at birth. In another case the child developed the eruption while being suckled by its mother, who was suffering from a septic affection.

Regular little epidemics of the disease have been observed to follow the practice of one physician or midwife.



Cultivation from the serum of the blebs has produced a golden yellow staphylococcus indistinguishable morphologically from the ordinary staphylococcus pyogenes aureus, but on inoculation into the skin of a healthy person it gave rise to a perfectly clear vesicle.

The pathological anatomy is very simple. The bulla is produced by the elevation of the horny layer, the stratum mucosum remaining almost perfect beneath. There is a slight œdema of the true skin and exudation round the vessels, but this not a marked feature.

*Diagnosis.*—This is usually not a matter of great difficulty, the chief eruption from which it is to be distinguished being the bullous eruption of congenital syphilis. The fact that the syphilide is always situated upon the summit of a well-marked infiltration, and practically invariably attacks the palms and soles, which are spared in pemphigus neonatorum with almost equal regularity, is generally sufficient, but the other symptoms of syphilis and the general state of the child's health would be additional factors.

*Treatment.*—This consists simply of protecting the skin with some bland and non-toxic antiseptic, either in the form of a powder, such as equal parts of powdered boric acid and starch, or if extensive excoriation be present the boric acid ointment of the Pharmacopœia is preferable. Great care should be taken to prevent undue loss of heat, and the possibility of the child's taking cold.

(2) PEMPHIGUS ACUTUS MALIGNUS.—For the knowledge of this disease we are chiefly indebted to the work of Pernet and Bulloch. The disease usually occurs in those who have to handle meat or skin, and hence is most common in butchers.

In some cases there has been a definite history of a small wound on the hand a month or two before the outbreak of the eruption. The eruption itself has no particular characteristics which would serve to distinguish it from a severe case of pemphigus vulgaris, but it is accompanied by severe constitutional disturbance. The symptoms are fever, albuminuria, vomiting, diarrhœa, and general exhaustion; in fact we have before us the clinical picture of a severe intoxication. The disease is generally fatal (75 per cent of Pernet's collected cases), and its duration varies from twenty-four hours to about three weeks.

As regards its etiology and pathology the fact that it occurs in those who are in frequent contact with the skin and flesh of animals, together with the frequent history of a precedent wound, would point to its originating in a local inoculation. Demme has found in the serum of the clear unruptured bullæ a diplococcus which grew in a rosette form on agar, and did not liquefy gelatine very readily, and this discovery has been confirmed by Bleibtreu, Bulloch, and others. The coccus was pathogenic to guinea-pigs when injected into the pleura, but did not give rise to any definite changes when injected subcutaneously.

The pathological anatomy does not differ from that of pemphigus vulgaris sufficiently to require separate description.

*Prognosis.*—From what has already been said it may be judged that the outlook is almost hopeless, roughly three-quarters of the cases ending fatally within a very short time.

*Treatment.*—This should be by the ordinary methods of combating cases of septicæmia. The diet should be liberal, and possibly alcohol may be needed. Of internal drugs quinine appears to have been of service in some cases. Local treatment should consist of treating the patient on a water bed (or if possible perhaps in the continuous bath) and dressing the skin with absorbent and antiseptic applications. These requirements are well supplied by Lassar's paste (R Zinci oxidi, pulv. amyli, āā ʒij., acidi salicylici, gr. x. ; paraffini mollis, ʒss.).

(3) PEMPHIGUS CHRONICUS VULGARIS.—The disease may begin either insidiously or suddenly. In the latter case it is usually accompanied by shivering, malaise, and fever, and these symptoms may be more or less continuous or frequently repeated throughout the course of the disease. The eruption consists of a crop of vesicles and bullæ, usually arising from apparently healthy skin, more rarely appearing on the site of a pre-existent erythema. The individual lesions vary in size from that of a small pea to



that of a hen's egg, or sometimes even larger. The eruption is usually generalised from the first, and the lesions may be either scattered without any ordinary arrangement, or they may show a tendency to occur in circular groups (*Pemphigus confertus*). The typical bulla when first formed is tense, hemispherical, or oval, and contains a clear yellowish fluid. After a short time, however, the contents become opalescent from the emigration of leucocytes, and the wall becomes flaccid from the evaporation and absorption of the fluid. In some cases a slight amount of blood may be effused into the blister in its early stage, giving a pink tinge to the contents. This condition is common, occurring usually in some of the lesions, and is to be carefully distinguished from those rare cases in which marked hæmorrhage takes place into all the bullæ (*P. hæmorrhagicus*). As the fluid of the bulla disappears, the roof flattens down and adheres by means of the albuminous remains of the former's contents to the floor, thus forming a scab. This falls off in a day or two, leaving a hyperæmic patch of skin, which may become pigmented later. The period of development and involution of the individual lesion usually lasts a week or ten days, but the disease is kept up by the appearance of new crops of bullæ. In some cases, probably from secondary infection, certain peculiar changes take place in the floor of the blister. Thus it may become covered with a grayish white false membrane (*P. diphtheriticus*), or may be attacked by a progressive gangrene (*P. gangrænosus*). Both of these sub-varieties are extremely fatal.

The course of the disease varies greatly, and the attack may either pass off within a couple of months or may be prolonged, with remissions and exacerbations for years. In mild cases the general health is not much disturbed,—soreness, burning, and, more rarely, itching being the chief symptoms; but if the rash is very severe, symptoms of exhaustion from pain, sleeplessness, and loss of heat and fluid, soon show themselves, and the patient finally dies from lung trouble, gastro-enteritis, or other intercurrent disease. Between these two extremes all varieties in chronicity are found, and even in cases which apparently recover in a short time relapses are common.

The chief complications of ordinary pemphigus are the implication of the mucous membranes of the mouth, vagina, and conjunctiva (for a description of pemphigus conjunctivæ see vol. ii. p. 362), and nephritis, the last-named being of very ominous significance.

(4) PEMPHIGUS FOLIACEUS.—This form may either commence as such from the first, or may supervene in a rebellious case of ordinary pemphigus. It is characterised by the appearance of ill-formed, flaccid bullæ with semi-purulent contents, or sometimes the epidermis is merely detached without being raised, so that it is hardly correct to speak of bullæ at all. Instead of drying up into a scab, as was described under simple pemphigus, the lesion spreads peripherally after the rupture of the covering, and no fresh, sound epidermis is formed from the floor of the original lesion. Thus the eruption becomes in time almost or quite universal, and the body is covered with shreds of partially detached epidermis, moist, red, exuding patches and brownish crusts. At this period infections become common, and boils and abscesses are of frequent occurrence. The patient develops a peculiar sweetish and most terribly nauseous odour, which is probably due to the decomposition of the albuminous discharge. In this stage vesicles are with difficulty even if at all recognisable. The whole epidermis appears to have become so rotten that the roof of any developing blister becomes broken at once.



All the epidermic appendages become involved in the disease, the hair falls out, and the nails become broken and frequently lost. The mucous membranes are also attacked, so that swallowing becomes a matter of difficulty, and the patient, by this time intensely emaciated, lies almost immovable, the slightest alteration of position causing fresh tears and cracks in the epidermis, and being accompanied by acute pain. Fever is constantly present, sleep and appetite are almost completely lost, and the patient finally succumbs to diarrhoea or pulmonary congestion.

(5) PEMPHIGUS VEGETANS.—This disease was originally described by Neumann in 1876, similar cases having been previously looked upon as syphilitic.

At the present time opinion is somewhat divided as to its nature, Unna preferring to separate it entirely from pemphigus under the name of Erythema bullosum vegetans, while others still believe it to be merely a variety of pemphigus.

The disease is an extremely rare one, about thirty undoubted cases only having been reported.

It begins usually in the mouth, and may be limited to this region for some time (one month in one of Herxheimer's cases). Later, bullæ which do not differ very strikingly from those of ordinary pemphigus, begin to make their appearance. There is, however, a peculiarity of distribution noticeable, in that the lesions seem to have a special predilection for the axillæ, genitals, and flexures of the thighs. There seems to be some doubt as to the appearance of the primitive lesion, Unna stating that it begins as "a red spot the size of a threepenny bit to a shilling, which may extend in one or two days to the size of the palm of the hand. It has sharply-margined, erysipelas-like borders, and there appear in the centre large thin-walled blebs, which soon dry up spontaneously into crusts." Kaposi also described them as beginning upon a red base in the form of lentil-sized vesicles, and says that in the first day or two the base resembles a wheel. Crocker, Herxheimer, and others, however, find that the bullæ have no special characteristics at first.

After the bullæ or vesicles are once formed they rapidly become cloudy, and usually dry up or burst in a day or two. The floor of the blister does not then form a new horny layer, but remains moist, red, and weeping, or may become covered with a brownish crust.

Meanwhile the process of vesicle formation spreads serpigiously at the edge of the former bulla, so that in time huge areas of skin become denuded of their supra-papillary epithelium. These denuded areas then begin to take on active growth, shared in both by the epidermis and papillæ, so that the characteristic condyloma-like structure is produced. This process is naturally most marked in the flexures, and the skin in these regions is constantly exuding a mucoid fluid with an extremely nauseating odour.

The hair becomes thin, and the nails brittle and often lost. Owing to the progressive implication of the mouth, nose, and pharynx, the ability of the patient to swallow nourishment is seriously interfered with. Emaciation from this cause, as well as from pain, sleeplessness, and chronic septic poisoning, becomes therefore extreme, and the patient almost invariably dies from exhaustion, nephritis, or lung trouble. Herxheimer has called attention to the presence of muscular tremors even during rest as symptoms of this and other severe forms of pemphigus.

*Etiology and Pathology.*—As has been already pointed out, there is little doubt that both varieties of acute pemphigus are of an infective nature. In the other forms little is known about the cause. The disease seems to affect both sexes



about equally, but is more frequent in children and in young adults than in people of more advanced age. Shock to the nervous system and severe chills seem to have an influence in its production, one case of *P. vegetans* having begun with multiple onychia after fishing in ice-cold water. The fact that bullous affections not infrequently occur in the course of nervous disease has led to the theory that pemphigus is itself a disease of nervous origin, and Schwimmer found degeneration of Goll's column in one case, and infiltration round the vessels of the posterior horn and posterior root in three cases. Petrini, Mott, and others have found degeneration of the peripheral nerves. On the other hand, careful post-mortem examinations have in numerous cases revealed no disease whatever of the central or peripheral nervous system.

The search for organisms has, up to the present, given no positive result in the chronic cases. The blood and the serum of the bullæ have been found to contain an abnormally large percentage of eosinophile cells, but since this condition has been found associated with several other diseases it has lost to a large degree the interest which was at first attached to it.

The anatomy of the lesion is still a matter of some uncertainty, some observers maintaining that the bulla is formed by the separation of the horny layer from the stratum mucosum, others that the entire suprapapillary epithelium is raised from the corium. The probability is that the separation does not always take place at the same level; but in the lesions which I have had the opportunity of examining the separation has taken place between the basal layer of the epithelium and the tops of the papillæ. The other morbid conditions found are of too general a nature to be useful in affording further knowledge of the nature of the disease, such as dilatation of the superficial blood and lymphatic vessels, œdema of the papillæ, and slight infiltration of round cells around the vessels. In pemphigus foliaceus these changes are far more marked than in ordinary chronic pemphigus, while in pemphigus vegetans there is superadded an enormous overgrowth of the epithelium and a marked infiltration of corium with round cells.

The diagnosis of ordinary well-marked pemphigus usually presents no difficulties, though in some cases it may be impossible to distinguish it at first from *Dermatitis herpetiformis* (*q.v.*).

The chief distinguishing points from this disease are: (1) The absence of herpetiform grouping; (2) The uniformity of the lesion, the bullæ springing from normal or slightly hyperæmic skin, no papules, wheals, or erythematous patches preceding the lesion. It should be noted that the bullæ of pemphigus *leave* hyperæmic patches, but these are easily distinguished. From erythema multiforme the same points, with the addition of the more acute course of the erythema and the tendency to concentric arrangement, should be sufficient. From the bullous syphilide of infants the diagnosis has already been given, and the same points, namely, the presence of the marked infiltration in the floor of the bulla, should render mistakes unlikely. The bullous lesion in leprosy may be distinguished by the presence of areas of anæsthesia and the thickening of the large subcutaneous nerve trunks.

In epidermolysis bullosa (described below) the definite action of trauma in producing the blisters, the fact that the disease is congenital in origin, and the common presence of scarring are the chief differential points.

*Prognosis.*—In simple uncomplicated pemphigus the prognosis appears to be good for the individual attack, but is doubtful for the ultimate health of the patient. The eruption is usually much more amenable to treatment in young patients, and it appears that permanent cures are more frequent in early life. The presence of marked constitutional symptoms, the affection of mucous membranes, and the presence of muscular tremors, should suggest a cautious prognosis. In pemphigus foliaceus and vegetans the disease has been up to the present so almost uniformly fatal, that the prognosis must be admitted to be hopeless.

*Treatment.*—The patient should in all cases be placed upon a water-bed, unless facilities are present for treating him by the continuous bath method, and this would only be necessary in very severe cases. Diet should be liberal, but alcohol is not usually indicated, unless signs of circulatory failure are present.

There appear to be several drugs which are extremely useful in alleviating



suffering, and thus indirectly benefiting the patient, but arsenic stands out pre-eminent for its power of controlling the outbreaks of the eruption.

It frequently needs to be given in full doses up to fifteen minims of the liquor three times a day. Some authorities prefer to give the drug in pill form, but this appears to possess no advantages, while it is certainly more difficult to ensure the free dilution which is so important a factor in its administration. Next in value to arsenic come quinine and opium, both of which occasionally exercise a distinctly curative effect. Lastly, phenacetin and its congeners are occasionally useful in relieving the distress and procuring sleep.

Locally, the bullæ may be punctured as soon as they become tense, and the fluid allowed to drain away. Every care should be taken to keep all excoriated places from becoming infected, while at the same time strong antiseptics must not be applied on account of their irritating qualities, and also because of the danger of absorption. In most cases Lassar's zinc paste (given above) meets all requirements admirably, though some patients are more comfortable when dusted over with a powder containing 25 per cent of boric acid. Whichever of these applications be used, it must be applied thickly, so as to soak up all discharge at once.

**EPIDERMOLYSIS BULLOSA HEREDITARIA.**—This disease was separated off from true pemphigus in 1882 by Goldscheider. The name was, however, suggested by Köbner, and is not perfectly satisfactory, inasmuch as the disease, although frequently a family affection, is by no means always hereditary. The condition is usually noticed soon after birth, and not infrequently during the first washing of the child. The skin "comes off" with the friction of the towel, leaving a raw, weeping surface beneath. A day or two later it is noticed that blisters are occasionally present, though in the early period of the child's existence any bullæ formed are so quickly ruptured that they may escape observation. As the child grows older and begins to move about it is found that the slightest friction or pressure is sufficient to cause a bulla to rise, often with blood-stained contents. The child is henceforth almost constantly exhibiting large areas of skin denuded of its surface epithelium, and, since the epidermis is lost to a great depth, scarring of a superficial nature is the rule, and contiguous surfaces not uncommonly heal together. Thus in a series of three cases in one family of children all had acquired more or less webbed fingers and toes. Some parts of the body are, of course, more exposed to trauma than others, and consequently show the ravages of the disease more markedly than others. Thus in addition to the webbing of the fingers already mentioned, a condition only seen in severe cases, the nails are almost invariably broken, deformed, or totally lost. Besides the skin the mucous membranes may also be affected, and in more than one case it has been noted that the patient has suffered from hæmatemesis, with the vomiting of œsophageal casts. The teeth are in most cases lost very early in life, owing to an abnormal brittleness and tendency to caries.

*Etiology and Pathology.*—Little is known on this subject. The disease occurs very distinctly both as a family affection and in sporadic cases. Descent from the father or mother has been observed, but it is more usual for several members of the same generation to be affected. As regards the pathology, some observers claim to have discovered lesions in the superficial blood-vessels, while others have described a peculiarly unstable condition of the basal layer of the epithelium. I am inclined to think that in neither case was the anatomical abnormality found the basis of the disease, but rather the effect of bygone traumatic lesions. Thus in endeavouring to obtain an apparently normal piece of skin from a patient, I found that the whole



surface was so studded with fine, small, superficial scars that this was impossible, and in the piece that I eventually examined I found only a slight mantle of cells round the vessels, especially marked under a spot where the epidermis was markedly thinned by diminution of the stratum mucosum, and which corresponded to one of the fine scars. Such a condition I have never seen in any other disease, and can only describe it as an epidermic scar.

The treatment of this affection is naturally unavailing, and all that can be done is to see that the child is as far as possible protected, and that later on it never has food at too high a temperature or too highly spiced. If lesions occur on the fingers, careful bandaging must be resorted to to prevent any healing together.

There appears to be some tendency to outgrow the affection, but I am unaware of any recorded case in which the condition has entirely ceased.

The diagnosis is simple. A pemphigoid eruption beginning in the earliest period of life, unaccompanied by any deviation from health, and called forth by trifling injury, makes a clinical picture which can be mistaken for no other disease.

**LITERATURE.**—The bibliography of Pemphigus is so enormous that only a few chief references are given, those being chosen which contain important work and a good list of further references :—

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## Penis, Surgical Affections of the.

(See also BLADDER, CIRCUMCISION, VENEREAL SORES.)

### INFLAMMATORY AFFECTIONS

*BALANITIS* or *Balano-posthitis*.—This consists in inflammation of the glans-penis and inner surface of the prepuce. It is common in dirty people, and is predisposed to by a long foreskin, in which secretions decompose and set up inflammation. It has been divided into venereal, catarrhal, diphtheritic, gouty, herpetic, and diabetic. The venereal form is due to retained discharges of gonorrhœa and venereal sores. The catarrhal form is due to decomposition of retained smegma in phimosis. Gouty balanoposthitis consists of red and raw patches, more on the glans than the prepuce. It is probably a form of gouty eczema. The patches may ulcerate and cause secondary phimosis. Diabetic balanoposthitis is similar to the moist eczema found in the female genitals.

*Cavernitis*.—This is an inflammation of the erectile tissues, generally due to erysipelas, cellulitis, or injury. A form of *chronic induration* of the erectile tissue occurs in gouty subjects, and consists in local patches of induration behind the glans or in front of the pubes. Ricord has described a similar condition after gonorrhœa and syphilis.

*Cellulitis* may occur in the penis from inflammation within the urethra or without. The former is due to gonorrhœa, or inflammation of the glands and lacunæ of the urethra; the latter to phimosis, balanitis, injury, etc.

*Elephantiasis* rarely attacks the penis alone.



*Erysipelas* may attack the penis, usually by extension from other parts.

*Gangrene* of the penis may occur after specific fevers, such as small-pox, typhoid, and typhus. Local causes may be phimosis, sloughing, balanitis, paraphimosis, phagedenic sores, or injury by rings, etc.

*Gout*.—Acute gout of the penis was described by Paget, occurring as a gouty urethritis, with swelling and heat of the penis. Chronic interstitial cavernitis and gouty balanitis are other conditions due to gout.

*Gummata* occasionally occur in the penis, but are rare.

*Herpes Progenitalis*.—This consists in the development of vesicles on an inflamed base, either on the glans or the inner side of the prepuce, occasionally on the outer surface of the prepuce. The vesicles dry up in a few days, leaving small scabs. If irritated the affection may suppurate. Sometimes it is preceded by severe neuralgic pains. The disease has a tendency to recur. The diagnosis of herpes from chancre is seldom difficult unless the vesicles have suppurated. It must be remembered, however, that herpes may furnish a breach of surface through which the syphilitic virus may penetrate and develop a chancre at the site of the herpes. Treatment consists in cleanliness and simple dusting powder.

*Lymphangitis* occurs as a complication of gonorrhœa and venereal sores.

*Neuralgia* of the penis occurs in gout, according to Paget.

*Tuberculous ulcers* may occur on the penis, sometimes from inoculation of a circumcision wound.

### NEW GROWTHS

1. *Simple*.—The chief of these are nævi, cartilage, and bone, sebaceous cysts, horns, and papillomata. The occurrence of cartilage and bone is rare, and in most cases due to calcification and gouty thickening. In some cases true osseous tissue has been found in the septum between the corpora cavernosa. Velpeau has recorded a case of a bony prolongation from the pubes, forming a true os penis. Sebaceous cysts sometimes occur in a long foreskin. Horns may arise from a wart or a sebaceous cyst. They are of two forms: one a flat plate, the other a projection, which may be an inch or more long. They should be removed by excision, as they may give rise to epithelioma. Papillomata may occur as simple warts on the penis or as venereal warts on the glans and foreskin. Venereal warts are thought by some to be due to irritation from venereal or leucorrhœal discharges, by others to be independent of them. In the female they may attain a large size, forming pedunculated masses round the vulva. They should be excised either by the knife or thermo-cautery.

*Malignant Tumours; Epithelioma*.—According to Paget, this forms one per cent of all cancers. It begins on the glans and prepuce, and may be superficial or deep. It usually arises as a warty excrescence which ulcerates, infiltrates the adjacent tissues, and usually assumes a cauliflower form of growth. Another form begins as a deep-seated lump of induration. Epithelioma may also arise in a chancreous ulcer, or in the healed scar of a chancre. Microscopically epithelioma of the penis is of the squamous form. Implication of the lymphatic glands is a grave complication, but visceral metastases are rare. It is rare before forty-five years of age. Predisposing causes are phimosis, venereal sores, gouty balanitis, and injuries. *Diagnosis*.—This is most important to make in the pre-cancerous stage, and all doubtful warty growths should be removed early. A foul discharge between the papillæ and a fixed and indurated base suggests malignant disease. From indurated chancre the diagnosis is usually easy, since the chancre is an ulcerated induration, while the epithelioma is an



indurated ulceration. The deep-seated form of epithelioma may, however, be difficult to diagnose.

*Sarcoma.*—This may occur as a primary sarcoma of the erectile tissues or secondary to sarcoma of the testicle. The sarcoma is usually round and spindle celled. A few cases of melanotic sarcoma have been recorded. The only treatment is extirpation of the penis.

#### CHIEF OPERATIONS ON THE PENIS

*Amputation of the Penis.*—This is indicated in cases of epithelioma and sarcoma. The operation may be partial or complete.

*Partial Amputation.*—This is best done by a flap operation. A narrow-bladed knife is entered well behind the disease, between the corpus spongiosum and the corpora cavernosa, and a short inferior flap is cut. From this the urethra is dissected out. A dorsal flap is cut from the dorsum and sides of the penis, and the corpora cavernosa divided at the point of transfixion. After stopping hæmorrhage the dorsal flap is punctured and the urethra drawn through it, slit up, and stitched. The flaps are then united. This method secures a skin covering for the corpora cavernosa.

*Total Extirpation.*—This operation is required for severe cases extending back to the scrotum. The scrotum is split along the raphe and the corpus spongiosum dissected out, cut through, and enough left to bring out at the perineum. By a blunt dissector the crura and capsule are detached on each side from the pubic arch. The suspensory ligament is divided and the dorsal arteries secured. The cut end of the corpus spongiosum is slit up and stitched to the posterior part of the scrotal incision and the wound closed. Any enlarged glands should be removed at the same time. Wheelhouse advises castration after amputation of the penis, as the testicles are a source of annoyance.

#### Other Conditions of the Penis

*Chordee.*—This occurs as a complication of urethritis, and consists of painful erections at night. The penis is twisted or bent downwards. This is due to effusion of inflammatory products into the corpus spongiosum, causing unequal distension of the penis during erection. The best *treatment* is a camphor pill at bed-time, or one containing opium, camphor, and belladonna. A morphia or cocaine suppository may also be given.

*Preputial Calculi.*—These may originate in the bladder or *in situ*. In the latter case they are due to smegma impregnated with lime salts or to stagnated urine in a long prepuce. Vesical calculi may be arrested in a phimosed prepuce.

*Priapism.*—This is a form of continuous erection without sexual desire. It may be complete or incomplete. It is caused by injury during coitus or excessive indulgence, giving rise to extravasation of blood into the cavernous tissue. It may also follow injury to the cervical or upper dorsal cord. In children a temporary priapism is caused by phimosis, vesical calculus, or ascarides. If due to extravasation of blood the condition may last several weeks. *Treatment* is not satisfactory; purgatives, tartar emetic, mercury, and iodides have little effect. Incisions have been tried with success.

LITERATURE.—W. H. A. JACOBSON. *Diseases of the Male Generative Organs.*—*Idem.* *The Operations of Surgery.*—BERKELEY HILL and COOPER. *Syphilis and Local Contagious Disorders*



**Peptonised Foods.** See INVALID FEEDING.

## Pericardium, Diseases of.

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See also HEART and MEDIASTINUM.

**THE PERICARDIUM.**—The pericardium is a fibro-serous sac which envelops the heart. The fibrous portion is pyramidal in shape, and at its base is firmly attached to the central tendon of the diaphragm and the adjoining muscular substance; above it is continued as a tubular prolongation on to the root of the aorta and pulmonary artery, and is gradually lost in the connective tissue of their external coats. The serous membrane lining the sac, termed the parietal layer of pericardium, passes up to the root of the great vessels, which it envelops in a common sheath for an inch to an inch and a half from their origin, and is thence reflected on to the surface of the heart, which it closely invests, constituting the visceral layer of pericardium. Laterally and anteriorly, save for a small triangular area in front termed the anterior mediastinum, the pericardium is in contact with the pleura; posteriorly it is in relation with the œsophagus, aorta, trachea, and the root of the left lung. The phrenic nerves pass down, one on either side of the sac.

### DISEASES OF THE PERICARDIUM

**PERICARDITIS.**—Pericarditis, or inflammation of the pericardium, may be sero-fibrinous or suppurative. The former is by far the most common variety, and may be acute or chronic. As a result of the latter, or of relapsing pericarditis, adhesions may form between the parietal and visceral layers of pericardium, constituting the condition known as “adherent pericardium.”

**ACUTE FIBRINOUS OR SERO-FIBRINOUS PERICARDITIS.** — *Etiology.* — *Rheumatism* is by far the most common cause of pericarditis. In childhood and early adolescence, from the ages of eight to twenty-five, rheumatism is especially liable to attack the heart, setting up pericarditis and endocarditis, with myocarditis in varying degree. In later life, while the articular manifestations of rheumatism are usually more severe, the heart is less frequently attacked.

*Chorea.*—Pericarditis occurring in association with chorea may be regarded as of rheumatic origin.

*The Rôle played by Micro-organisms in Pericarditis.*—The belief has long been held that acute rheumatism is a disease of microbic origin, and that pericarditis and endocarditis are local manifestations of the activity of the rheumatic micro-organisms. Of recent years various observers have turned their attention to this question. In 1886 Mantle wrote a paper on the subject. In 1881 Achalme isolated and described a short bacillus which he considered pathogenic of rheumatism, and his views were supported by Thiroloix and Bettencourt. In 1898 Triboulet isolated a diplococcus from the blood of a patient suffering from acute rheumatism, and by inoculating it into a rabbit produced endocarditis. In 1899 Wasserman obtained from the blood and pericardial fluid of a rheumatic patient pure cultures of a diplococcus which, inoculated into rabbits, gave rise to pyrexia with polyarthrititis, but no valvulitis. In 1900 Dr. Paine and Dr. Poynton brought forward important and apparently conclusive evidence that rheumatic fever is due to a minute diplo-



coccus which they consider identical with that described by Wasserman and Triboulet. This organism they have demonstrated in the fluid in the pericardium and in joints, and also in the tissues of patients suffering from rheumatic fever. They have also isolated it from the pericardial fluid, and after growing it as a pure culture on suitable media and in pericardial fluid have inoculated it into rabbits, reproducing in them polyarthritides, pericarditis, endocarditis, myocarditis, and pleurisy. It would appear, therefore, that we are justified in regarding pericarditis as one of the local manifestations of the activity of the rheumatic diplococcus, and must look upon it not as a complication of rheumatism, but as an integral part of the disease.

*Pleurisy and Pneumonia.*—Pericarditis may occur in association with pleurisy and pneumonia, possibly by direct extension of the inflammatory process, but more probably through the agency of the specific micro-organisms which were the cause of the primary disease. In view of the recent researches by Dr. Payne and Dr. Poynton mentioned above, it seems probable that in rheumatic subjects, when pleurisy is associated with pericarditis, both may be due to the rheumatic diplococcus. The same may hold good of pneumonia in some cases, but it has been shown that the pneumococcus may set up pericarditis, and when pneumonia is the primary affection the pneumococcus is usually found to be the cause also of the pericarditis if a bacteriological investigation is made.

*The Acute Specific Diseases.*—Acute pericarditis may occur as a complication of scarlet fever, variola, erysipelas; rarely of measles, typhus, enteric fever, and malaria.

*Bright's Disease.*—Pericarditis is met with as a complication of Bright's disease most commonly in association with granular kidney or subacute parenchymatous nephritis.

*Traumatism.*—Injuries to the pericardium by a fractured rib, or by a foreign body, such as a needle or fish-bone lodged in the œsophagus, or gunshot and punctured wounds, may set up pericarditis.

*Tuberculosis and Malignant Disease.*—Tubercular pericarditis is usually secondary to pulmonary tuberculosis, tubercular disease of the mediastinal glands. Malignant disease and new growths may occasionally invade the pericardium and set up pericarditis.

*Suppurative* pericarditis will be discussed later.

*Morbid Anatomy.*—In the early stages the pericardium becomes hyperæmic and congested, and assumes a dull red colour instead of its normal glistening appearance. Exudation of serum and leucocytes takes place from the congested vessels, and soon both visceral and parietal layers of pericardium become coated in patches or throughout their surface with a thick layer of yellow sticky lymph. From the friction together of the two inflamed surfaces the lymph coating them acquires an irregular, ragged, or honeycomb appearance. The inflammatory lymph on the surface of the pericardium may be reabsorbed, but frequently becomes vascularised and organised into fibrous tissue, gluing together the two layers of pericardium where they come into contact, and giving rise to partial or universal adhesion between the heart and pericardium.

*Effusion* may take place into the pericardial sac to a varying extent. Usually it is small in amount, but exceptionally it may be very large, as much as two pints having been recorded.

In rheumatic cases a large effusion is uncommon. As a rule in fatal cases in children there is little excess of fluid found in the pericardial sac post-mortem, but the two layers of pericardium are coated with thick yellow lymph.

The effusion consists of a yellowish serous fluid in which leucocytes, shreds of fibrin, and endothelial cells are present in varying quantity. The fluid is usually clear, but may be turbid if there is much cellular exudation, and may contain flakes of lymph. Sometimes it is blood-stained, and in scurvy may consist almost entirely of blood.

*The Myocardial Changes.*—These are of importance, as the myocardium seldom escapes damage in cases of pericarditis. They are described in detail elsewhere



(v. "HEART"), but it may be as well to mention here that they consist of degenerative and fatty changes in the muscle fibres with foci of small round-celled infiltration in the interstitial tissue.

*General Description of the Course of the Disease.*—The only certain evidence of the onset of pericarditis is a to-and-fro pericardial friction sound heard on auscultation, caused by the rubbing together of the inflamed surfaces of the pericardium at each beat of the heart. Before this becomes manifest certain premonitory symptoms, as a rule, make their appearance. These are: acceleration of the pulse; excited or tremulous action of the heart; increase in the respiration rate, with dyspnoea; rise of temperature; and præcordial pain in varying degree. In children restlessness, night terrors, slight delirium on waking, and irregular action of the heart may sometimes be present in addition in acute cases. There is always some dilatation of the heart, sometimes appreciable before the appearance of the friction rub, usually pronounced in the later stages in severe cases.

The pericardial rub persists for a varying time—from a day or two to a month or more, according to the severity and chronicity of the attack. The disappearance of the rub may indicate: (1) The subsidence of the attack; (2) Effusion into the pericardial sac; (3) The formation of adhesions between the two layers of pericardium.

These points are all discussed at length later on. The physical signs and symptoms of pericarditis will now be considered in detail.

**PHYSICAL SIGNS.**—*Pericardial Friction Sound.*—This, the characteristic sign of pericarditis, is caused by the rubbing together of the two inflamed surfaces of pericardium. It is a soft scratching sound, heard on auscultation, somewhat resembling the noise made by scraping a piece of rough paper with the finger-nail. In chronic cases it may to a certain extent lose this soft scratching character and become harsh and rough. It appears to be very superficial, and is modified by pressure with the stethoscope, being rendered harsher and more distinct, or almost extinguished, according to the degree of pressure exerted. Sometimes change of position, *e.g.* from the recumbent to the erect posture, will render the rub more distinct when it is not well heard.

*Site of the Rub.*—The pericardial rub may be heard at any point over the præcordial area. It is localised at the outset, and is usually first heard at the base of the heart or over the lower end of the sternum; but frequently it is first apparent at the apex. It may remain localised to a small area, and then disappear, or may extend so as to be audible over the whole of the front of the heart, and when there is much dilatation may be heard for some distance to the right of the sternum.

*Rhythm of the Rub.*—The friction rub is of a to-and-fro character, corresponding more or less to the movements of the heart in systole and diastole. Its rhythm varies somewhat according to its situation. When heard at the apex of the heart it may be a single systolic scratch synchronous with the apex beat, or a to-and-fro rub. When heard over the lower end of the sternum and between the sternum and apex it has a see-saw rhythm which is not exactly synchronous with the heart sounds, and is readily distinguishable from them. When heard at the base, over the auricles, it has a triple cantering rhythm which is very characteristic.

*Friction fremitus* is best heard by placing the flat of the hand over the præcordial area. It is by no means always present or appreciable, but is usually most distinct where the pericardial rub is loudest and roughest. It is a curious scratching sensation of feeble intensity, and appears to be very



superficial; it has not the vibratory character of the thrill associated with an endocardial murmur.

*The Heart.*—The heart frequently appears to be excited out of all proportion to the severity of the attack, and the pulse is almost invariably accelerated. There may be a tumultuous heaving, wavy impulse over the whole præcordial area, and auricular pulsation is sometimes seen in the second and third intercostal spaces in children, while epigastric pulsation may be present as well. These phenomena are usually most marked in prolonged or recurrent attacks, when the cardiac dilatation is pronounced.

*The Pulse.*—The most constant and characteristic change in the pulse is acceleration of the rhythm which is often pronounced before the friction rub is audible. Sometimes in children it is irregular, or intermits a beat at irregular intervals, but this is very rare in adults in the earlier stages. The pulse wave is not at first noticeably affected, but in severe and prolonged attacks it becomes sudden, short, and ill-sustained. The pulse is then one of low tension and diastolicity is well marked.

*Increase in the Area of Cardiac Dulness.*—One of the earliest and most characteristic physical signs in acute pericarditis is the increase in the area of cardiac dulness, which can be readily made out on percussion. It is more rapid and extensive in proportion to the severity and acuteness of the attack, and is also very pronounced in the later stages of subacute or relapsing pericarditis. It is most marked in children, in whom also it can more easily be mapped out than in adults, as the chest wall is thin, and there are no emphysematous changes in the lungs. The greatest increase is usually to the right, and the dulness may extend to an inch or more beyond the right margin of the sternum, and upwards as far as the second rib.

Sibson apparently took it for granted that increase in the area of cardiac dulness indicated pericardial effusion, and this view has been maintained by many authors since. In a certain small proportion of cases this undoubtedly holds good, but there is much evidence to show that in by far the greater number of cases of rheumatic pericarditis this increase in dulness indicates not effusion, but dilatation of the heart, more especially of the right auricle and ventricle.

In 1895, in a monograph on "Adherent Pericardium," I described a series of cases tending to prove this; in two of these paracentesis pericardii was performed in the belief that the rapid and extensive increase in the cardiac dulness indicated effusion. In neither instance was fluid found, but the needle punctured the dilated right ventricle, fortunately without ill results.

In 1898 Dr. Lees and Dr. Poynton, in a paper, read before the Medico-Chirurgical Society, on "Acute Dilatation of the Heart in Rheumatism and Chorea," also drew attention to this point. From an analysis of 150 cases of fatal rheumatic heart disease in children they found that in only 12 was there marked excess of fluid in the pericardium, but that cardiac dilatation was the rule.

Clinically it is a common experience in subacute cases, when the area of cardiac dulness is very much enlarged, extending for an inch or more to the right of the sternum, to hear a pericardial rub at one or more points over the dull area. This seems incompatible with the presence of fluid, and in these cases, at the post-mortem, no fluid is found, but a dilated heart, sometimes with the pericardium partially adherent.

*Signs of Effusion.*—Disappearance of the friction rub with progressive and extensive increase in the area of cardiac dulness are usually regarded



as indicative of effusion into the pericardial sac; but, as has been stated above, increase in the area of cardiac dulness in a large proportion of cases indicates, not effusion, but dilatation of the heart, and the disappearance of the friction rub may be due, not to fluid, but to the formation of adhesions between the two layers of pericardium. It is by no means an easy matter to determine, by percussion alone, when effusion takes place. The outline of the cardiac dulness in effusion is described as being pyramidal or pyriform in shape, with the base or widest part below. Sibson in his treatise on Pericarditis gave a series of diagrams based on percussion, illustrating the shape assumed by the pericardial sac in the various stages of effusion, but it is doubtful how far these are of practical value for differential diagnosis, as they are not confirmed by post-mortem examination, and it is possible that cardiac dilatation may have been mistaken for effusion in many instances.

It is not safe, therefore, to dogmatise as to the exact shape and extent of the area of cardiac dulness in the earlier stages of effusion, but it is generally admitted that it increases in all dimensions and tends eventually to assume a pyriform outline. Dr. F. Roberts also points out that in extensive effusion dulness can be made out to the left considerably beyond the apex beat.

The transition from the dull area to the resonant lung is sudden and well marked, and the extent and shape of the cardiac dulness will vary somewhat with change of position of the patient, being increased in its upper part when the patient sits up and leans forward. This, however, is not a safe test, as it may produce fatal syncope.

Rotch and Ewart state that in effusion there is dulness in the fifth right intercostal space, due to accumulation of fluid in a pouch in the right corner of the pericardial sac, and that this dulness is not present in simple dilatation. I have, however, in several instances found dulness in this position in cases in which post-mortem no fluid was found, but a dilated heart with the pericardium adherent.

*Change in the Position of the Apex Beat.*—Sibson and other observers have stated that effusion causes displacement of the apex beat upwards and outwards, but there is much diversity of opinion on this point, and, moreover, in extensive effusion the apex beat is usually imperceptible.

*Progressive enfeeblement of the apex beat* from day to day, and its eventual disappearance, together with increasing weakness and distance of the heart sounds as heard at the apex, in association with progressive and rapid increase of the area of cardiac dulness, are strongly suggestive of pericardial effusion. Confirmatory evidence will be afforded if at the same time there is marked exacerbation of the symptoms, such as præcordial oppression, cyanosis, fulness of the veins of the neck, a small, feeble, and irregular pulse, urgent dyspnœa, so that the patient cannot lie down, but finds most relief in sitting up and leaning forwards over a bed-table.

*Symptoms.*—The symptoms are partly local, due to the inflammation of the pericardium, and partly constitutional, due to a general toxæmia from the poisons evolved by the rheumatic micro-organisms.

Pain in the præcordial region, increase in the pulse and respiration rate, dyspnœa, and rise of temperature, are symptoms well marked at the onset of acute pericarditis, and usually precede the appearance of the pericardial friction rub. In children nervous symptoms may be present in addition, such as crying out and starting in sleep, restlessness, and slight delirium on waking. In the subacute and relapsing type common in children there may be little or no pain, and very slight pyrexia and dyspnœa on exertion only; in short, the symptoms may be so slight, that the child



is able to go about during the early days of an attack, or even for the greater part of its course, with disastrous ulterior results. Pronounced anæmia, the presence of rheumatic nodules, and the history of previous attacks of rheumatism, are usually distinguishing features of this class of cases.

The face is usually pale, but may be flushed, the expression is anxious and distressed, and respiration is hurried. The temperature ranges between  $100^{\circ}$  and  $103^{\circ}$ , and is usually irregular; in the insidious and subacute attacks in children it is often very little above normal, but is always somewhat irregular. There may occasionally be some pain or difficulty in swallowing from the proximity of the pericardium to the œsophagus, if the posterior aspect is inflamed or if there is much effusion. Dyspnœa is usually a prominent feature, and may become very severe, not only in cases of effusion, but when there is great cardiac dilatation and the pericardium is partially or wholly adherent to the heart. The patient may have to be propped up in a sitting posture, and find most comfort in leaning forward over a bed-board.

*Vomiting* is sometimes present and is always a serious symptom; not infrequently it ushers in the closing scene.

Nervous symptoms may predominate in children, and, as pointed out by Sturges, there may be great restlessness, with delirium and sleeplessness. In exceptional cases there may be pronounced mental change, a condition of maniacal excitement, or of melancholia, sometimes with hallucinations. The former I have seen twice in a case of pericarditis complicated by severe chorea, the latter once in a case of relapsing pericarditis. Both were in children, and both proved fatal.

In the later stages the patient may sometimes lapse into a semicomatose state, passing motions and urine into the bed.

*Course of the Disease and Terminations.*—The disease, as a rule, runs a somewhat different course in adults and in children. In the former it is usually acute and is ushered in by severe symptoms. In the latter it is frequently subacute and insidious in its onset, and results very frequently, after a protracted course, in the formation of adhesions between the two layers of pericardium.

In a favourable case, though the early symptoms may be severe, the friction rub persists for a few days only, the area of cardiac dulness does not become very greatly enlarged, rapidly decreases to normal again when the friction rub has disappeared, and convalescence is soon established.

Very severe attacks may prove fatal within a few days after the pericardial rub is first heard, but this is unusual unless there is some additional complication such as chorea or pneumonia. In such cases there is usually rapid and extreme enlargement of the area of cardiac dulness, severe dyspnœa, high temperature, rapid pulse, and sometimes vomiting. In two such cases of which I have seen the post-mortem examination there was very little excess of pericardial fluid, only a little lymph on the surface of the pericardium which was intensely congested, but the dilatation of the heart was extreme. Possibly in such cases the severity of the inflammation may cause paresis of the cardiac muscle analogous to that which occurs in the muscular coats of the intestine in peritonitis.

In the subacute type of pericarditis common in rheumatic children, as first pointed out by Cheadle, the onset is usually insidious and the symptoms are ill marked, and when the child is first brought to a hospital by its parents a pericardial rub and mitral systolic murmur may already be present. The friction rub persists on and off for some days or weeks, the area of cardiac dulness becomes greatly enlarged, and never regains its



normal limits, and eventually, when the pericardial rub disappears, the pericardium is left partially or universally adherent to the heart. After convalescence is established the child frequently remains pale, anæmic, thin, and short of breath, and the heart hampered by adhesions, and with its muscle damaged by inflammation, remains permanently crippled.

If the inflammation is very protracted, or repeated attacks come in close succession, the cardiac dilatation may become extreme, the liver enlarged, and dropsy supervene, the patient eventually dying with all the symptoms of right ventricle failure. It must be borne in mind that the myocardium is almost invariably, and the valves are frequently, attacked by the inflammatory process of which pericarditis is a part, and that the ultimate issue of the illness and prognosis for the future largely depends on the degree in which the cardiac muscle is damaged. For, not only does the dilatation of the heart due to myocarditis favour the formation of adhesions between the two layers of pericardium during convalescence, but the eventual replacement of damaged muscle fibres by cicatricial fibrous tissue leaves the heart permanently weaker.

*Diagnosis.*—Till the characteristic friction rub is heard, a diagnosis of pericarditis cannot be made with any degree of certainty. There are, however, as a rule, in acute cases premonitory symptoms, such as acceleration of pulse, excited action of the heart, rise of temperature, dyspnœa, and perhaps præcordial pain or oppression, which enable us to anticipate the appearance of the pericardial friction rub. These symptoms may, at first sight, seem to be merely exacerbations of those already present from rheumatism, scarlet fever, or pneumonia, with which pericarditis may be associated; but if we bear in mind the possibility of cardiac complications and examine the heart daily, we are not likely to mistake their significance or to miss the first definite signs of pericarditis.

It is otherwise with the subacute attacks of pericarditis common in children, which are often so insidious in their onset that not only may there be no premonitory symptoms, but the pericardial rub may have been present some days, and much damage to the heart have resulted before the child is seen to be ill and medical advice sought.

In Bright's disease and tuberculosis also, the onset of pericarditis may be very insidious. Attention is drawn to this later on where the subject is more fully discussed.

*Differential Diagnosis.*—The pericardial friction rub is so characteristic that it is seldom likely to be mistaken for other sounds. Difficulty may, however, arise, when from friction between the parietal layer of pericardium and the adjoining pleura in a case of pleurisy, the rhythm is of a to-and-fro character corresponding to the beats of the heart. A pleuro-pericardial rub arising from this cause is usually intensified in deep expiration, and rendered less distinct in deep inspiration, and is modified by the respiratory movements. It is, as a rule, only heard at the left border of the heart, and not over the sternum or to the right of it. Moreover, as the pleurisy will seldom be confined to the neighbourhood of the heart, a respiratory friction rub will be heard in the adjoining area in the axilla.

In obscure cases it will be necessary to bear in mind the possibility of the somewhat rare affection, acute mediastinitis, of which an account is given, vol. vii. p. 362.

*Endocardial murmurs* differ so completely in their character from pericardial friction sounds that confusion will seldom arise. They have, as a rule, a focus of intensity, are conducted in certain directions, and have a definite relation to the first or second sounds of the heart, which they may partially



or entirely replace. They are not appreciably modified by pressure with the stethoscope, and though they may sometimes be accompanied by a thrill, and be rough and vibratory, they never assume the superficial scratching character of the pericardial friction rub.

*Effusion.*—The differential diagnosis between effusion and cardiac dilatation has already been discussed. Difficulty in diagnosis between effusion and aneurysm or mediastinal growth is scarcely likely to arise if the history of the case is known.

*Prognosis.*—Pericarditis is always a disease of considerable gravity. In acute attacks associated with rheumatism or pneumonia death may take place within a few days, but this is not of common occurrence. In the majority of cases the immediate prognosis, so far as life is concerned, is favourable, but the liability to repeated attacks, the damage done to the heart by myocarditis, and the formation of adhesions between the heart and pericardium, render the ultimate chances of long life unfavourable in a large proportion of rheumatic cases. The prognosis will to a great extent depend on the degree in which the myocardium is affected by the inflammatory process. If the attack of pericarditis is short, though severe, and the heart does not become greatly dilated, the changes in the muscle will probably be slight, and the heart may rapidly recover afterwards. If the inflammation is protracted, or repeated attacks occur in close succession, the myocardium will seldom escape serious damage; there will be great dilatation of the heart, from which it is unable to recover for some considerable time, and the pericardium not infrequently becomes adherent to the dilated heart, rendering its complete recovery impossible. Moreover, as in the process of repair, fibrous tissue takes the place of the damaged muscle, the contractile power of the heart is permanently weakened.

Children and young adults on whom rheumatism has set its mark in the shape of the characteristic "nodules" are especially liable to repeated and prolonged attacks of carditis or inflammation of pericardium, myocardium, and endocardium. They seldom grow up to maturity, or if they do, it is with hearts so crippled that long life is scarcely possible.

*Treatment.*—Treatment should be both local and constitutional. Of local applications the ice-bags applied over the pericardial area are the most useful, as they relieve pain and seem to have a steadying effect on an excited heart; they are usually well borne and liked by the patient. Leeches are advocated by some at the onset of an acute attack, but it is doubtful if they are of any real service, and they may upset the patient, and thereby do more harm than good. Blisters are useful in the later stages of a prolonged attack or in chronic pericarditis.

The administration of salicylates, which in the acute articular rheumatism of adults are given with such marked beneficial results, seem to have no effect in arresting the inflammation, and may do harm if pushed. If the pain is severe and there is great restlessness and dyspnoea, opium or morphia may be employed with marked benefit. Digitalis and cardiac tonics of the same character should not be given during the acute stage of pericarditis, but if stimulants are required, brandy, ammonia, ether, and strychnine or nux vomica may be given. Quinine given in an effervescing mixture with bicarbonate of soda or potash is the most generally satisfactory and useful medicine. The administration of iodide of potassium is advocated, but is of doubtful service. Nothing in severe cases seems to arrest or exert any controlling influence in the course of cardiac inflammation in children on whom rheumatism has got a firm hold.

It is, therefore, of importance that any indications of danger threatening



the heart should be recognised as early as possible, and due precautions taken. If a suspicion of rheumatism is aroused by pains in the limbs or joints in a child who comes of a rheumatic stock, or who has previously suffered from rheumatism, the patient should be kept under observation and the heart examined from day to day. If rheumatic nodules are present danger to the heart is imminent, and the child should be carefully looked after and guarded from exposure to cold and exertion of any kind, while the heart should be examined, and the morning and evening temperature and pulse-rate should be noted daily.

*Effusion.*—If extensive effusion into the pericardial sac takes place, its removal by paracentesis may become necessary, and if the symptoms are severe it should not be long delayed, as death from syncope is liable to occur. It is, however, frequently a very difficult matter to determine whether the increase of cardiac dulness and the symptoms of cardiac embarrassment are due to effusion or to cardiac dilatation, and not infrequently the dilated right ventricle has been punctured by accident in the absence of supposed effusion. The best site for paracentesis is in the fifth left intercostal space, about 1 inch or  $1\frac{1}{2}$  inches from the sternal margin. Some advocate that it should be made in the fifth right space or in the angle between the xiphoid cartilage and costal margins. A small aspirating needle should be used, and previous to its insertion an incision should be made through the skin down to the intercostal muscle, so that any bulging of the space can be noted and the needle introduced without force and in any direction required.

The above description applies to the type of pericarditis which is met with in association with rheumatism, and it will be necessary here to briefly allude to the form it may assume in Bright's disease and tuberculosis.

*In Bright's disease* pericarditis occurs most commonly as a complication of subacute parenchymatous nephritis or of the chronic interstitial variety. Statistics vary as to its frequency, from 14 to 3 per cent, according to different authors. It is possible that this discrepancy may have arisen from classifying cases of hydropericardium and pericarditis due to intercurrent diseases, such as pneumonia and rheumatism, with those due to toxic influences the direct result of Bright's disease.

The onset of pericarditis in Bright's disease occurring, as it usually does, in debilitated subjects, may be very insidious and unattended by any marked symptoms of local reaction.

There may be little or no rise of temperature, no præcordial pain, and little beyond increase in the pulse-rate and slight exacerbation of pre-existing symptoms, such as dyspnoea and restlessness due to uræmia, to call attention to its presence. It may be accompanied by pleurisy, and it is often very difficult to distinguish between a pleuro-pericardial rub and a true pericardial friction sound.

It is important, therefore, that the heart should be examined as a matter of routine, or the onset of pericarditis may easily escape notice.

The prognosis is always very grave and the treatment must be directed to the primary disease.

*Tubercular pericarditis* usually occurs as a secondary infection, in association with tuberculosis of the lungs or of the bronchial or mediastinal glands. It may, however, be a primary affection, and the only tubercular lesion, according to Virchow, Joffroy, Cornil, and others, but this is very rare. Tubercles on the pericardium are also present sometimes in acute miliary tuberculosis.



Tubercular pericarditis may be acute or chronic. The former is most commonly due to direct invasion by an adjacent tubercular gland, and is usually attended by effusion. The pericardium may become thickened, or the effusion become purulent from infection by a caseating gland, so that the tubercular origin of the disease may escape notice without careful histological investigation.

In the chronic variety the onset is insidious, and there is usually little or no effusion. The two layers of pericardium become thickened and adherent, and may contain caseous masses or recent tubercles. The history of the case and the physical signs will be those of chronic mediastinitis or adherent pericardium.

Osler points out that tubercular pericarditis often escapes notice during life owing to its insidious nature, and is frequently not detected post-mortem, as the thickened and adherent pericardium is attributed to other causes. He recognises four groups of tubercular pericarditis:—

1. When the condition is latent, and the disease is only discovered post-mortem in persons who have died from pulmonary tuberculosis or other causes.

2. Cases in which the symptoms are those of cardiac embarrassment due to adherent pericardium from chronic tubercular pericarditis.

3. Cases of acute pericarditis attended by effusion, serous, hæmorrhagic, or purulent in character, in which there may be no suspicion of the tubercular origin of the trouble.

4. A group where the clinical picture is that of acute general tuberculosis. (See *American Journal of the Medical Sciences*, Jan. 1893.)

#### ADHERENT PERICARDIUM

By the term “adherent pericardium” is implied the presence of adhesions between the two layers of pericardium as a result of antecedent pericarditis.

*Morbid Anatomy.*—The adhesions may be limited to fibrous bands stretching across the pericardial sac at one or more points, or they may be universal, so that the pericardial cavity is entirely obliterated. In the case of recent adhesions, or of lymph undergoing organisation into fibrous tissue, the two layers of pericardium can be separated, and their surface will present a rough honeycomb appearance; but if the adhesions are of old standing they are tough and fibrous, and the pericardium cannot be stripped from the heart without tearing its muscular substance. Adhesions may also exist between the pericardium and chest wall, or adjoining pleura and lung, as a result of so-called mediastino-pericarditis. See article on “Mediastinum,” vol. vii. p. 363.

In association with adherent pericardium there is commonly some fibroid change in the heart walls due to replacement of fibrous tissue of muscle fibres destroyed by previous inflammation.

*Physical Signs.*—The physical signs differ according as the adhesions exist only between the two layers of pericardium, or between this and the chest wall or adjoining pleura as well. In the latter case they are more numerous and distinctive, and those that may be present are the following:—

*Systolic depression* of one or more intercostal spaces to the left of the sternum, or of the lower end of the sternum itself and the adjoining costal cartilages, which may be caused by the heart dragging on them at each systole through the pericardial adhesions. Systolic recession of spaces alone is, however, not a trustworthy indication, as it may be due to atmospheric pressure when the heart is hypertrophied and the patient is a child



with yielding chest walls. Care should be taken not to mistake auricular pulsation, sometimes seen in the left intercostal spaces, for systolic retraction.

*Systolic retraction* of the lower portions of the posterior or lateral walls of the thorax may indicate the existence of an adherent pericardium when the heart is enlarged, and the pericardium is adherent to the chest wall as well as to the heart. The explanation of such retraction seems to be that the portion of the diaphragm to which the pericardium is adherent is dragged upwards at each systole of the heart, so that the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and retracted.

*Fixation of the apex* beat, so that it does not alter its position in respiration or in change of posture, is an important sign when present.

*Systolic recession* over the site of the apex beat, or a diastolic shock on palpation over this area, point to the presence of pericardial adhesions between the apex of the heart and the chest wall.

*Impeded descent of the diaphragm* in inspiration, as shown by deficient movement of the epigastric region, may indicate the presence of abnormal adhesions between the heart and diaphragm, and is more marked if there are adhesions between the chest wall and diaphragm in addition.

*The area of cardiac dulness* will be abnormally large, and will remain unchanged in respiration when there are extensive adhesions between the pericardium and the chest wall, as the thin layer of lung tissue normally overlapping the base of the heart will be pushed aside, or more probably be involved in the adhesions and collapsed.

*Enlargement of the Heart.*—It is common, but by no means constant, to find the heart considerably enlarged when the pericardium is considerably adherent, even in the absence of valvular lesion or other obvious cause to account for it. It seems probable that such enlargement may be due in some measure to pericardial adhesions, as follows:—The heart becomes dilated during an attack of pericarditis, and the muscular fibres are also weakened by inflammation. Before these recover and the heart contracts again to its normal size, the pericardium becomes adherent and fixes it in a condition of dilatation. Subsequently the heart undergoes some hypertrophy.

*Diastolic collapse* of cervical veins, held by Friedreich to be of diagnostic value when accompanied by systolic retraction of spaces, is seldom seen.

*Symptoms.*—These are not in themselves characteristic, but are such as may arise from embarrassment of the heart, especially of the right ventricle, shortness of breath, palpitation, præcordial oppression, ascites, and dropsy.

*Diagnosis.*—The physical signs and symptoms, few of which may be present, are often of themselves insufficient to allow of a diagnosis being made, or of the condition being suspected in the absence of a history of pericarditis; but valuable help may be derived by careful consideration of the question, "Are the symptoms such as the physical signs would lead one to expect, or are they severe out of all proportion?" If the latter, adherent pericardium must be thought of as a possible cause of the unexpected breakdown of the right ventricle when there is no exciting cause, such as undue strain or exertion, or kidney disease, or lung trouble to account for it. For the right ventricle, with its thinner walls, is most seriously hampered by pericardial adhesions, and in association with these there is frequently some substitution of fibrous tissue for muscle damaged by previous inflammation, so that an unexpected breakdown may readily occur. When a suspicion of adherent pericardium is thus aroused confirmatory evidence in the form of



physical signs of this condition must be carefully sought for. Frequently, however, in spite of all care a certain diagnosis is impossible.

In cases of pericarditis which can be kept under observation for some time after the attack there is less difficulty in detecting the presence of adhesions. The indications which would lead one to suspect that the pericardium was becoming adherent are:—

Prolongation of the attack of pericarditis evidenced by the persistence of a harsh friction rub at various points over the pericardial area for some weeks, together with marked increase in the area of cardiac dulness, which remains permanently after all evidence of pericarditis has disappeared. This will in many instances, but not invariably, be due to the pericardium becoming adherent to a dilated heart; and later other physical signs of adherent pericardium may develop.

*Prognosis.*—When the heart remains normal in size after the subsidence of pericarditis, and there are no adhesions between the pericardium and chest wall, the universal adherence of the pericardium to the heart does not appear to materially shorten life. When the pericardium becomes adherent to a dilated heart and also to the chest wall the prognosis is more serious. When adherent pericardium is a complication of valvular disease it is liable to prove fatal at an earlier date by so hampering the right ventricle as to interfere with its recovery when compensation breaks down. It must not be forgotten that the degree in which the myocardium has been damaged at the time of the attack of pericarditis is an important factor in prognosis.

*Treatment.*—The discovery of adherent pericardium is important from the point of view of treatment, not because anything can be done to remedy or remove adhesions when found, but because it will be necessary to impose additional restrictions on the patient, so that no undue risks may be run.

**SUPPURATIVE PERICARDITIS.**—Purulent pericarditis may occur as part of a general pyæmic or septicæmic condition, or in association with empyæma, abscess of the lung, suppuration of mediastinal or cervical glands, or of other adjacent structures. Sometimes no apparent cause can be found.

The inflammatory process is almost invariably septic from the onset, due to the invasion of pyogenic micro-organisms, and is attended by effusion of pus into the pericardium. Seldom does a serous effusion of rheumatic or tubercular origin become purulent.

The onset of the disease is, as a rule, insidious, and the discovery of pus in the pericardium is often not made till the post-mortem examination.

*Physical Signs.*—There is seldom any definite friction rub to announce the onset of the attack. The physical signs present will be those of pericardial effusion, which have already been discussed. Chief of these is increase in the area of cardiac dulness, and the difficulty in distinguishing whether this be due to cardiac dilatation or effusion will be intensified when no friction rub has been heard to suggest the possibility of effusion. Furthermore, it not infrequently happens that the area of cardiac dulness is encroached upon by, or runs into, a large dull area due to empyæma. Feebleness or loss of the apex beat and weak or distant heart-sounds may be noted, but will not be of great value, unless the case has been watched from the outset, so that a standard of comparison could be formed. There may, occasionally, be œdema over the præcordial region.

*Symptoms.*—The temperature is usually that characteristic of some septic affection, but may sometimes be little above normal throughout. Rigors seldom occur unless as part of a general septicæmia, except in the somewhat rare event of a serous pericardial effusion becoming purulent. Pain is absent as a rule, but there may be a feeling of oppression in the



præcordial region. The pulse-rate and respiration are accelerated, and there is usually dyspnoea, especially on movement.

*Diagnosis.*—The diagnosis presents many difficulties, and is frequently not made during life. In the absence of an antecedent friction rub effusion may not be suspected, and the intermittent pyrexia may be attributed to an empyæma, or suppuration elsewhere than in the pericardium.

If effusion is suspected an exploring-needle may be employed. The skin should be incised parallel to the ribs, as described under paracentesis pericardii, and the intercostal muscles divided before the needle is inserted into the pericardium, so that any bulging of the space may be noted, and the needle be accurately directed.

*Prognosis.*—The prognosis is always very serious, but there are reasonable grounds for hope in cases where the suppuration is not part of a general septic infection, but is a sequela of an empyæma or other localised abscess.

The earlier the diagnosis is made and surgical interference is sought, the better will be the chance of recovery. The percentage of recoveries is, however, very small.

*Treatment.*—Abscess in the pericardium must be treated like any other abscess. It should be opened and drained under antiseptic precautions as soon as the diagnosis of suppuration is made.

**HYDROPERICARDIUM.**—By hydropericardium is meant effusion of serous fluid into the pericardial sac, as the result, not of inflammation, but of passive dropsical transudation. A certain amount of clear serous fluid, varying from half an ounce to three ounces, is commonly found in the pericardial sac post-mortem, the effusion probably taking place during the act of dying.

During life hydropericardium, or effusion of such degree as to be clinically recognisable, occurs most commonly as part of a general dropsical transudation from whatever cause, and is therefore most frequently associated with Bright's disease or morbus cordis. It is much less common than effusion into other serous cavities, and when present is a late feature in the disease, and is gradual in onset. It is said also to occur as the result of mechanical obstruction, to the return of blood from the pericardial and cardiac veins from some local cause, such as pressure by mediastinal tumours, enlarged glands, or fibrous adhesions, but this must be very exceptional.

A more rapid and acute serous effusion into the pericardium may sometimes take place in acute nephritis.

The fluid consists of serum, and is usually pale yellow in colour and clear, but may be turbid from the presence of desquamated epithelium and leucocytes, and is sometimes blood-stained.

*Symptoms and Physical Signs and Diagnosis.*—The symptoms are increasing dyspnoea and præcordial oppression with enfeeblement of the pulse, but as they will usually be present to a varying degree, as a result of the pre-existing disease with which the condition of hydropericardium is associated, they are not characteristic.

The physical signs are those of pericardial effusion, increase of the area of cardiac dulness, with progressive enfeeblement of the apex beat and weakness of the heart-sounds, but as there is no antecedent friction rub to call attention to the possibility of effusion, it may readily escape notice in the earlier stages. The diagnosis cannot be made unless the effusion amounts to several ounces, and when there is effusion into one or both pleural cavities as well it is very difficult, and may be impossible.

*Treatment.*—Practically the treatment is that of the original disease to



which the general dropsy and hydropericardium are due. Relief may occasionally be afforded by aspiration of the pericardium when the diagnosis of effusion can be made with certainty; but it is rarely employed or called for, as the relief can but be temporary, and does not long delay the fatal result.

**PNEUMOPERICARDIUM.**—The presence of gas in the pericardial sac is rare. It may be due to penetrating wounds of the pericardium by a sharp instrument or a fractured rib, but is more commonly the result of ulceration, by which a communication is established between the pericardial sac and an air-containing cavity. Thus, an old phthisical cavity, an abscess in the lung, a pneumothorax, a subphrenic abscess that has made its way through the diaphragm, may open into the pericardium, or a communication may be established with the œsophagus by ulceration, giving rise in each instance to pneumopericardium. Pus may find its way into the pericardium, with the air giving rise to pyo-pneumopericardium, or blood in the case of punctured wounds, when the condition is termed hæmo-pneumopericardium. The entrance of gas into the pericardium is always attended by inflammation, most commonly of a septic character, so that pyo-pneumopericardium is the usual result. The gases most frequently found in addition to air are sulphuretted hydrogen and carbon disulphide, but these are only found as a result of putrefaction.

The gas may distend the pericardium, and always rises to the highest part of the cavity with change of posture of the patient.

*Physical Signs.*—Uniform bulging of the præcordial area may be noted. The apex beat is usually absent or feeble, but can sometimes be felt when the patient bends forwards.

*On percussion* the note varies with change of position, being dull over the fluid and tympanitic or high pitched over the air-containing cavity. The relations of the gas and fluid will be remarkably altered by changes of posture, and can be readily made out by percussion.

*On auscultation* the heart-sounds acquire a peculiar metallic character, and are unusually loud and clear, so that they may sometimes be heard at some distance off, and are a source of annoyance to the patient himself. Splashing sounds, and metallic tinkling, and a bell note with coins can usually be heard.

*Treatment.*—As pneumopericardium is usually a complication of some grave disease, treatment must vary accordingly. Aspiration may be required to remove the gas or fluid, or possibly free incision may be necessary to allow of the escape of pus.

**NEW GROWTHS OF THE PERICARDIUM.**—Primary cancer of the pericardium is scarcely ever met with, but two cases of carcinoma are recorded by Bernheim and one of sarcoma by Sir Wm. Broadbent.

*Secondary carcinoma* is also very rare, and when found is usually due to direct invasion of the pericardium by a new growth in the mediastinal or bronchial glands, lungs, or adjacent structures, very exceptionally to metastasis from a distant growth.

It is, as a rule, accompanied by pericarditis with effusion usually hæmorrhagic in character.

The condition can only be suspected if a pericardial friction rub is heard when carcinoma in the neighbourhood of the pericardium has been diagnosed. It may be impossible to diagnose effusion when it occurs if there is already a large area of dulness due to a new growth in the anterior mediastinum.

*Hydatid of the Pericardium.*—Cases of this affection are on record, but it is so rare that it must be regarded as a pathological curiosity.



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**Perihepatitis.** See LIVER.

**Perinephritis.** See KIDNEY.

**Perimetry.** See VISION.

**Perinæum.** See PELVIC FLOOR.

**Periosteum.** See BONE.

## Peritoneum.

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### I. ANATOMY

THE peritoneum is the delicate connective tissue membrane which lines the pelvis and the abdominal cavity, and which is reflected from their walls over the viscera which they contain.

Originally it was continuous with the pleuræ and the pericardium, the whole membrane then forming the lining of the undivided body cavity or cœlom. At a later stage the membrane covering the thoracic viscera is separated from the peritoneum and undergoes further specialisation. It must, however, be remembered that the connection between the peritoneum and the serous sacs in the thorax remains exceedingly close throughout life. By means of their lymphatics they are probably in direct connection with one another. Their structure is the same, the pathological changes which they undergo are allied, and their broad anatomical arrangement is identical. Their anatomical similarity might at first sight be overlooked as it is obscured by the complexity of the peritoneum, which depends upon its huge area of surface and the number of organs with which it comes into relation. Not only are the organs very numerous, but in the course of development many of them undergo changes in form and position, which materially modify the peritoneum over them. Even with the help afforded by the study of the simpler forms of peritoneum found in animals and in the embryo, it is hard to understand the anatomy in the adult, while, without the assistance so obtained, the task might well appear impossible. Like the other serous sacs, the peritoneum is composed of two parts, a parietal lining the walls of the cavity, and a visceral which is reflected over the organs. The two are continuous, and between them a potential space exists, called the peritoneal cavity. In health there is no actual space, so that the term peritoneal "cavity" applied to the normal condition is misleading.



In passing to the viscera folds of membrane are formed. They contain a delicate groundwork of connective tissue, which lies between the layers of serous membrane and affords support to the vessels and nerves which run in them.

In the female the peritoneal cavity is not closed, because the Fallopian tubes open directly into it. This curious arrangement distinguishes the peritoneal from all the other serous sacs, and is of clinical and scientific interest. To the physician it explains the liability of women to develop pelvic sepsis, and it provides the histologist with a unique example of the junction of a mucous with a serous membrane. For descriptive purposes, however, the peritoneum in the female, as well as in the male, may be considered a closed sac which is tucked around and between the different organs.

The primitive peritoneum is nothing more than the differentiated lining of the abdominal cavity. By the forward growth of the gut it is separated into a visceral and parietal portion. The alimentary canal is at first a median structure lying along the vertebral wall of the abdominal cavity. It then greatly increases in length, the ends being fixed so that the lengthened central part falls forward, forming a loop. As the loop pushes its way towards the front of the abdominal cavity, it carries a layer of peritoneum before it. This layer is the visceral portion of the membrane, and ultimately forms the mesentery. Differentiation of the peritoneum naturally occurs *pari passu* with that of the alimentary canal, so that as stomach, small intestine, and large intestine are formed on the one hand, the mesogaster, mesentery, and mesorectum arise on the other. These special names given to the different parts of the visceral peritoneum increase the difficulty of the subject.

In the adult the peritoneal cavity consists of two diverticula, the greater and lesser peritoneal sacs. The greater is the one which is usually spoken of as the peritoneal cavity *par excellence*, and is the one which is opened in operations upon the intestines and the other abdominal viscera. The lesser sac, sometimes called the omental sac, lies behind and below the stomach, and extends downwards into the pocket enclosed by the folds of the great omentum. The two cavities communicate by a narrow opening, the foramen of Winslow, which lies beside the neck of the gall-bladder. It may be demonstrated by turning the stomach and omentum upwards and passing the finger along the neck of the gall-bladder. It is bounded in front by the free edge of the gastro-hepatic omentum, behind by the inferior vena cava, above by the liver, and below by the duodenum.

In the adult these two peritoneal sacs seem to be almost divided from each other, because the opening between them is so small and so hidden. In reality, however, they are nothing more than two pouches of one cavity. The smaller peritoneal sac is the upper and right half of the original abdominal cavity, and it only becomes shut off from the rest of the cavity during changes in development. If the changes which take place in the stomach are carefully followed, it will be easy to understand the way in which the lesser peritoneal sac becomes shut off from the greater.

Originally, as the upper end of the alimentary canal, the stomach is a median and vertically placed structure, slung between two folds of peritoneum, which pass forwards to the liver and back to the vertebral column. Presently it rotates, turning upon its right side, till in the end it lies at right angles to the long axis of the body, instead of parallel to it, so that what ultimately forms the small curvature is morphologically its anterior border, and the great curvature its posterior border, while the pylorus is the lower end. The two ends of the organ, the pylorus and the cardia,



retain very much their original relation to one another, as they are comparatively fixed, while there is great growth in the intervening muscular wall. The stomach, therefore, comes to form a pouch.

The fold of peritoneum which slings it to the spine (its mesentery or mesogaster) is naturally affected by the changes which it undergoes in shape and position, and instead of passing back to the spine as a straight fold, as it did with the primitive vertical stomach, it forms a pouch with its concavity looking upwards and to the right. The formation of this pouch divides the abdominal cavity into its two diverticula, the upper one being at this stage very small in comparison with the other. It is increased in size by the down-growth of the mesogaster to form the great omentum, and when these changes are effected, it constitutes the lesser peritoneal sac, which is thus seen to be nothing more than the upper and right part of the original peritoneal cavity shut off from the rest of the cavity by the curved mesogaster as it follows the rotating stomach.

The free edge of the gastro-hepatic omentum does not grow proportionately, and it remains as the anterior boundary of the foramen of Winslow.

The peritoneum does not cover all the organs equally. It entirely surrounds parts which, like the coils of small intestine, require free mobility, and it appears to enter into specially intimate relations with organs which possess marked powers of absorption and secretion, like the stomach, the pancreas being a possible exception. The relation of the peritoneum to individual organs is often of surgical importance. In lumbar colotomy the incision is planned to reach the uncovered posterior surface of the descending colon. In impermeable stricture of the urethra, supra-pubic puncture of the bladder may safely be performed directly above the pubes, because in distension of the bladder the reflection of peritoneum is carried 2 to 4 cm. above the symphysis pubis. The long mesentery and the free mobility of the small intestine explain why it occurs in such overwhelming proportion of cases of hernia. Hernia of the large intestine is relatively uncommon, although one variety, hernia of the cæcum, has been the subject of much discussion. It was at one time believed that the cæcum did not possess a complete peritoneal coat, it was thought that when it took part in a hernia the sac was incomplete. Investigation has, however, shown that as a rule this is not the case, but that in 90 per cent of cases the cæcum has an entire serous investment and even a mesentery of its own. In a small proportion of cases the posterior surface of the cæcum is not covered by peritoneum, and if hernia occurs in these cases, the relation of the gut to the sac may be very puzzling, and may cause mistakes of a serious nature. In some cases of this kind the cæcum lies in front of the sac, and has been mistaken for it and opened in operations for the relief of strangulated hernia. In a patient recently admitted to the Royal Free Hospital the reverse condition existed, the sac in his case lying in front of the cæcum. When the sac was opened it was found to be empty, while the gut lay behind it. The peritoneum over the anterior surface of the cæcum formed the posterior wall of the sac. At points of fixation along the alimentary canal peritoneal folds occur, forming fossæ. They are found at the ileo-jejunal flexure, about the cæcum, where Lockwood and Rolleston describe three, and in the neighbourhood of the sigmoid. Internal hernia may occur in connection with these folds, or with the lesser peritoneal sac. Hernia into the lesser peritoneal sac may occur either through the foramen of Winslow, or through the central part of the gastro-hepatic omentum, which is weaker than the rest, and is called the *pars flaccida*.

The attachment of the mesentery runs obliquely from above down, and divides the greater peritoneal cavity into lateral compartments, while the planes of mesentery running to the various coils of intestine lead to still further subdivision. This helps to prevent the general distribution of fluids in infectious invasions; for example, the ascending mesocolon in suppurative appendicitis often restricts the pus to the iliac fossa until adhesions have formed.

The loins form the lowest part of the peritoneal cavity, and in the recumbent position free fluid collects in them rather than in the pelvis—a point which should be remembered in draining the abdominal cavity.



## II. PHYSIOLOGY

The mechanical function of the peritoneum, as it is sometimes called, is the one which first strikes the observer. For a long time it was regarded as its only function.

Mechanically, the peritoneum acts like a joint. Free and frictionless movements of the viscera which it covers are the requirements demanded of it, and by its anatomical arrangement and by its smooth surface, which allows the viscera to glide freely over one another, they are admirably met.

It possesses, however, other functions which are no less important in the economy of the body. To properly appreciate them, it is necessary to study the peritoneum in relation to the rest of the lymphatic system of which it forms a part. It is the largest lymph space in the body, being, according to Wegner, equal in extent to the entire skin. It communicates freely with the neighbouring lymphatics, which can easily be injected from its cavity. There is a constant flow of lymph from the peritoneum through the lymphatic plexus in the diaphragm towards the thoracic duct, and so into the general circulation.

The peritoneum possesses almost incredible powers of absorption and secretion. In man they are evidenced in disease; in animals they have been tested experimentally. It is impossible to exaggerate their physiological importance. The fact that the absorptive power of the peritoneum is about ten times greater than that of the synovia of joints in itself, speaks for its power of natural resistance. It has been shown that, in the living animal, water injected into the peritoneal cavity is absorbed at the rate of 10 per cent of the body weight, and in the dead animal at 5 per cent of the body weight, per hour. If, on the other hand, hyper-tonic fluids, such as concentrated sugar solution, be used, its powers of secretion will be demonstrated, for a serous exudation takes place into the peritoneal cavity, amounting to 3·8 per cent of the body weight of the animal in an hour.

Probably in health fluid is constantly being exuded into the peritoneal cavity from the vessels which form so close a network over its surface, but as there is exact correspondence between absorption and exudation, no residual fluid collects.

The normal peritoneum is wet and glistening. It is like silk which has just been wrung out of water. It is believed that a number of factors contribute in maintaining the balance between the two processes of absorption and exudation. In disease it is disturbed, and the peritoneum either becomes too dry, or fluid accumulates, distending its cavity.

The vascular supply of the peritoneum is exceedingly rich, and for this reason clean wounds in the membrane heal remarkably well—better, indeed, than those of any other part of the body. Its nerve supply is also abundant, and the connections which are formed between its nerves and important centres, such as the solar plexus, are numerous and interesting.

By studying the anatomy of its nervous supply, their connections and their modes of origin, an explanation is obtained of many symptoms and reflex phenomena which are of common occurrence in abdominal disease or injury. The peritoneum is supplied with nerves from both the spinal and sympathetic systems. The connections between its spinal nerves and the solar plexus and the cardiac branches of the vagus explain the shock and cardiac inhibition which follow abdominal injuries as reflexes, and often form such a fatal group of symptoms. The skin of the anterior abdominal wall is supplied with cutaneous branches from the last seven dorsal nerves. These nerves also supply the lower intercostal muscles and the muscles of



the anterior abdominal parietes, and through the splanchnics they take an important part in the nervous supply of the peritoneum.

If the peritoneum is injured, as in peritonitis, rigidity of the muscles forming the front wall of the abdomen and hyperæsthesia of the skin follow naturally as reflexes from the anatomical nervous connections. The board-like rigidity of the muscles, the intense sensibility of the skin, which shrinks from the slightest pressure, and the shallow breathing, due to reflex affection of the intercostals, all serve a useful purpose in helping to keep the inflamed part at rest and free from further injury.

There has been some discussion as to whether the healthy peritoneum is or is not sensitive. Probably it is not. Opening the bowel in the second stage of colotomy is scarcely felt by the patient, and in cases in which opportunity has arisen of handling the peritoneum without an anæsthetic it has not apparently caused pain. A clean cut in it is painless, while the slightest degree of inflammation causes acute suffering.

The outer surface of the peritoneum consists of a network of elastic fibres mingled with lymphatic and vascular capillaries and nerves. The elastic fibres are important, as they endow it with distensibility, so making it a suitable covering for organs like the stomach and uterus, which vary in size from time to time.

Its structure must be studied from fresh preparations treated with a weak solution of silver nitrate.

Its inner surface is composed of flat pavement endothelial cells resting upon a basement membrane, and broken at intervals by apertures, stomata, and pseudo-stomata. The outlines of the cells show up as dark wavy lines in the silver nitrate specimens. They fit into one another by their sinuous margins. It is thought that the pseudo-stomata are formed by a heaping up of the intercellular cement at various points between the cells. They have no known physiological function. In his famous experiments upon the absorption of milk from the peritoneal cavity, Recklinghausen first described the stomata as the path through which absorption occurred. He regarded them as organised channels between the peritoneal cavity and the lymphatics. They are openings at the junction of several endothelial cells, and are lined with small cells which differ from the normal cells of the part in being granular and cubical. Solid particles suspended in fluid and injected into the peritoneal cavity rapidly pass into the connective tissue spaces and the lymphatics in the parietes, and until recently the stomata were universally regarded as the channels of exit through which they passed. Lately, however, Recklinghausen's conclusions have been disputed, and the existence of organised channels of communication between the peritoneal cavity and the sub-endothelial lymph spaces has been disputed. Until the matter is settled, stress must not be laid on stomata in discussing the mechanism of peritoneal absorption.

The path taken by fluid in its passage from the connective tissue spaces into the general circulation is also a point over which there has been much difference of opinion. The lymphatics used to be regarded as the main channel of absorption, aided only to an insignificant extent by the blood capillaries.

During the last few years some physiologists have attempted to minimise their importance, and to prove that the capillaries are the main path of absorption.

The following points have been brought forward in favour of lymphatic absorption :—

1. Anatomically, as the spaces open directly into the lymphatics, the connection between them is closer than that between the spaces and the capillaries.



2. The pressure in the lymphatic system is lower than that in the capillaries, and fluid always tends to flow in the line of diminishing pressure.

3. Ligature of the thoracic duct hinders absorption from the peritoneal cavity.

4. Substances absorbed from the peritoneal cavity are said to appear in the lymph, collected by a canula in the thoracic duct before they appear in the urine (blood).

Potassium ferro-cyanide was used in these experiments, and the urine and lymph collected from the bladder and thoracic duct respectively were examined at short intervals for the Prussian blue reaction.

Unfortunately experimenters have not reached the same conclusions. Meltzer always found that the reaction was given by the lymph before the urine, while Starling came to the opposite conclusion.

The arguments which have been used to establish the superior importance of the capillaries resolve themselves to a great extent into a negation of the points enumerated above.

It is said that—

1. Stress must not be laid upon the seeming continuity of the lymphatics and the tissue spaces, nor upon supposed conditions of pressure in the lymph and vascular capillaries, because our knowledge is not sufficiently trustworthy.

2. During the rapid absorption of peritoneal effusions the flow of lymph from the thoracic duct is not increased.

3. Under certain circumstances it has been conclusively shown that absorption by the blood-vessels can occur.

Defibrinated blood led repeatedly through the blood-vessels of an œdematous amputated limb becomes more watery. In the control experiment, which differed only in the tissues being healthy, the blood became less rather than more watery.

The factors which produce absorption have also led to much discussion. It has been said to occur from filtration, in the line of diminishing pressure, by osmosis, and by vital cell action.

Probably several factors act in combination, especially filtration and osmosis. It is known that absorption of fluids from the peritoneum varies inversely with the osmotic pressure of the fluid. Hypertonic solutions become at least isotonic, generally hypotonic before absorption.

Robinson sums up the functions of the peritoneum under the following headings. He says it is of use—

I. To prevent friction of moving viscera.

II. To anchor and support the viscera in their proper relations to each other.

III. To prevent invasion of infectious micro-organisms by throwing out barriers of exudation to protect wounded viscera.

IV. To furnish the viscera with an elastic covering.

V. To absorb and secrete fluid.

### III. PATHOLOGY

*General Remarks.*—The realm of non-bacterial peritonitis is daily becoming more restricted, and it is now believed that the vast majority of cases of peritonitis, if not all, are due to pathogenic bacteria. Since, however, it is now known that micro-organisms only act by virtue of their chemical products, the distinction between chemical and bacterial infection need not be so sharply defined as was once thought necessary.

Cold probably never acts as an actual excitant, but only as a predisposing agent, by lowering the power of resistance of the tissues. The susceptibility of the peritoneum varies in different animals within wide limits, and probably it also varies to some extent in man, some people being able to deal successfully with doses of poison to which others would succumb.

Certain parts of the membrane are much more sensitive than others. The area of the small intestine is much more vulnerable than that over the colon, while the parietal peritoneum, compared with the visceral, is remarkably tolerant. The power of resistance possessed by the outer parietal peritoneum



is illustrated by cases of vertebral abscess, in which the pus is in contact with it for long periods of time without inducing peritonitis or leading to toxæmia from absorption.

A chronically inflamed peritoneum appears to be somewhat more tolerant of a slight degree of sepsis than a healthy one. It stands operation better. To a certain extent it has become immune. The pelvic peritoneum in women is thicker and more opaque than in men. The mesentery in the adult is less translucent than in the child. The reason is probably the same in both cases. The peritoneum represents a territory in which there is a struggle throughout life between invading micro-organisms and the leucocytes which guard it, and which can be called up at any moment for purposes of defence. The records of past struggles are written in the changes which occur in the membrane as age advances, and in those which are found in parts specially liable to damage, like the pelvic peritoneum in women.

The peritoneum itself has considerable power of resistance, depending chiefly upon its absorptive apparatus. Experiments show that in health it can deal successfully with fairly large injections of pyogenic organisms, provided that no nutrient material is introduced with them. The bacteria are encapsuled or absorbed. If, however, blood serum or lymph is present as well, the bacteria multiply and septicæmia follows. The practical indication of this observation is the importance of keeping the peritoneal cavity as dry as possible during operations. Hands and instruments, after purification, should be thoroughly dried, and when the operation is finished all moisture and blood-clot should be removed, while in peritonitis, drainage rather than irrigation should be the line of treatment. Peritonitis always begins locally. It is rather more common in women than in men, the "points of attack" varying, however, in the two sexes. In women pelvic troubles and inflammation about the gall-bladder increase the total number of cases, while in men appendicitis and hernia more often occur. In both sexes perforation of the gastro-intestinal track is a comparatively common and very fatal cause.

Peritonitis in its essence may be considered a salutary process. If it did not occur the simplest laparotomy would almost inevitably prove fatal. A single micro-organism introduced into the abdominal cavity would find itself in a haven of comfort, in which, undisturbed, it could multiply and invade the body. As it is, the organisms which reach the peritoneal membrane are at once surrounded by millions of leucocytes, which pour from its vessels and attempt to engulf them. It is by the reaction of the tissues alone that the patient can be saved. If he dies, his symptoms are those of septicæmia, not of inflammation. He dies of the poisons elaborated by absorbed micro-organisms, not of local inflammation, as the term "death from peritonitis" implies. Death takes place because peritonitis fails to save life. It is always the sepsis which kills, while the inflammation is the attempt, and often a successful attempt, to save life. In the most rapidly fatal cases, where the patient dies with symptoms of profound poisoning, it is usual to find nothing or next to nothing visibly wrong with the peritoneum at the post-mortem examination. The leucocytes in these cases have been at once overpowered, and no traces are left after death of their ineffectual struggle.

*Morbid Anatomy.*—Tympanitis occurs early from reflex paralysis of the bowel. It is most marked in the small intestine. The distended coils are hyperæmic and patchy, with a dull and lustreless surface. The injection on the walls of the intestine is most visible along lines parallel to the line of attachment of the mesentery. The distended coils of gut lie



against each other, and the lines of injection represent the angles between adjacent coils, where the pressure is least. They are sometimes, for this reason, called suction-lines. Flakes of lymph also collect in this situation. They vary in thickness, and can at first be detached from the membrane lying under them.

The microscopic changes are the same as those enumerated by Metschnikoff in his classical description of inflammation. Dilatation and stasis occur in the small vessels, and there is an exudation of lymph and migration of leucocytes through the vessel walls into the surrounding spaces. The exudate consists of a net-work of fibrin with white blood corpuscles in its meshes. At first it forms milky flakes, which later may become gray or greenish. Membranous adhesions are formed between opposed surfaces, and are gradually drawn out by the movements of the viscera into cords and bands.

When the acute stage is over the inflammatory products are either entirely absorbed or become partly "organised" into scar tissue, which contracts in the usual way.

It is usual to describe three forms of peritonitis separately, although in practice they are constantly met in combination, especially in cases of tubercular peritonitis.

They are respectively—(1) Dry, adhesive or fibrinous peritonitis.

(2) Serous peritonitis.

(3) Suppurative peritonitis.

*Adhesive Peritonitis.*—The adhesions may be so soft as to be easily broken down by the finger, or tough enough to resist cutting by a knife. They may form the merest tags attached to organs, or they may bind all the abdominal contents into an inextricable mass. In the early stages, by helping localisation, they are of infinite service; later, when they contract, they cause all kinds of trouble by the pressure they exert on nerves and other structures, and by interfering with the proper movements of the viscera. Chronic cases, in which the deformity is extreme, are known as cases of peritonitis deformans. The omentum may be enormously thickened and rolled up, the mesentery is shortened, and all the organs firmly bound down, with resulting loss of mobility and pain.

*Serous Peritonitis.*—In the sero-fibrinous variety the quantity of fluid varies greatly in amount; according to Osler, from  $\frac{1}{2}$  litre to 30 or 40 litres. In simple acute peritonitis the quantity of serous exudation is generally small. In carcinomatous infiltration of the membrane and in tubercular peritonitis it may be considerable. It is clear or cloudy, according to the proportion of contained leucocytes. In some of the acute cases the distended vessels rupture, when a blood-stained exudation results, while occasionally a markedly hæmorrhagic effusion occurs in cancerous and tubercular peritonitis. If there is perforation of the alimentary tract, the contents of the stomach or gut are mixed with the fluid and alter its character.

*Purulent Peritonitis.*—A general purulent peritonitis is rare, although sero-purulent effusions, often very copious, may occur in general peritonitis. With intense general sepsis, death takes place very rapidly from absorption, usually before the tissues have had time to react completely with resultant pus formation. This, no doubt, is the reason why general purulent peritonitis is rare. Circumscribed abscesses, however, are very common in the outlying parts of the abdomen in connection with the parietal layer of the peritoneum and the colon; for example, about the appendix, in the vault of the diaphragm, forming the so-called subphrenic abscess, and in the pelvis.

*Bacteriology.*—A large number of organisms are associated with peritonitis, and in the majority of cases the infection must be regarded as a



mixed one. Pyogenic organisms head the list, streptococci being by far the most important. (Tavel and Lanz found them in three-fourths of their cases.) Staphylococci are much less virulent, and rarely occur except in combination with streptococci.

Streptococci are the active agents in cases of peritonitis arising from external infection, from perforative wounds of the abdominal wall, from contamination during operations, in puerperal septicæmia, and occasionally also in association with cutaneous erysipelas. The fact, however, that the superficial lymphatics of the abdominal wall do not communicate with the deep system of peritoneal lymphatics prevents this from being a common accident. The cases due to infection from the alimentary tract are of great interest. Peritonitis follows perforation of the alimentary canal, but it also occurs in cases in which, although the gut-wall is injured, no perforation has occurred; for example, in volvulus, strangulated hernia, and non-perforative appendicitis. Cultures made in the ordinary way from the inflammatory exudation in these cases almost invariably give pure growths of the *bacillus coli communis*, or at any rate show growths in which colonies of this bacillus preponderate. The bacillus has been cultivated in this way from the fluid in the sac of a strangulated hernia, and from the peritoneal exudation in cases of non-perforative peritonitis.

Hawkins prepared stained sections of the appendix wall in cases of non-perforative appendicitis, which had nevertheless caused fatal general peritonitis, and which he included under the heading of infective appendicitis. He showed that bacilli were present on the peritoneal surface of the wall, instead of being restricted to the inner surface of the gut as they normally are. He believed that they were *bacilli coli communis*.

The burning question arises as to the part played by the ordinary intestinal bacteria, especially the *bacillus coli communis*, which stands as the prototype of the great colon group of bacilli in the causation of peritonitis. This bacillus was discovered in 1885, and at first it was described as a harmless inmate of the intestine, present throughout life, and entirely free from pathogenic properties. Then gradually evidence was amassed against it, in the shape of the facts cited above, and almost all cases of peritonitis arising from injury to the gut, with or without perforation, were laid to its account. It was believed, under certain conditions, to acquire great virulence, with the power of penetrating the wall of the gut, when its inner surface in any way became injured and its vitality lessened.

This theory has until lately been universally held. The objection to it is that it is a little fanciful, and that the extreme readiness with which the *bacillus coli communis* grows may lead to the most deceptive conclusions as to the frequency of its presence and its etiological importance. So much is this the case that recent observers hesitate before they allow it anything like the importance which its constant presence in the ordinary culture tubes, often in pure culture, seems to indicate.

The old theory, it must be admitted, is cut somewhat to fit the facts, to act as a cloak for ignorance; and moreover, recent work tends to show that, by special methods adopted to exclude air, anærobic organisms of the pyogenic class can be cultivated, and probably are mainly responsible for many cases of peritonitis which have previously been ascribed to the *bacillus coli communis*, because by the ordinary methods of cultivation they have been killed off, while the colon bacilli have flourished.

Dr. Veillon of Paris has recently made the most important contributions to this subject. His conclusions are principally based upon the results obtained from the study of twenty-two cases of appendicitis.



He believes that, in causing gangrenous and foetid inflammations, a group of strictly anaerobic bacteria play a role of prepondering, if not of exclusive importance, but that, as they require special methods of cultivation, they have hitherto escaped observation. Several years ago Veillon observed that in certain cases of suppuration there is a marked difference between the microscopic appearance of freshly-stained specimens of the pus and the results of culture.

In other conditions the results of immediate microscopic examination tally with those obtained from culture; that is to say, the same cocci are seen in the field of the microscope and in the colonies grown upon the agar-tubes after inoculation with the pus. In osteomyelitis they are found by both methods to be *staphylococcus pyogenes aureus*.

The cases in which the results of culture differed from the appearance of the slides were noticed to be those in which the inflammation was most intense, and the process foetid or gangrenous in nature. In the appendix many of them belonged to Hawkins' class of "infective appendicitis." In some of these cases, while the field in the fresh specimen was seen to be swarming with micro-organisms, hardly any growth occurred in the inoculated tubes; the colonies which developed in them were very few in number, and they only represented a small proportion of the forms seen in the freshly-prepared slides. The *staphylococcus* or *streptococcus*, with members of the colon group, the *bacillus coli communis* or the *proteus vulgaris*, developed in these cases, while other germs which had previously been seen on the slide did not grow upon the tube. Why, in these cases, was it that only a small proportion of the germs could be grown?

Before Veillon brought forward his hypothesis that the majority of these bacteria failed to grow because they were anaerobes, they were believed to be dead or at least dying before inoculation.

This explanation was unsatisfactory, because it was unlikely that specially virulent forms of inflammation should be caused by dead or dying bacteria. Veillon's hypothesis at last afforded a rational explanation of facts. He taught that, far from being moribund, the organisms in these cases were intensely active and virulent, and that if they were supplied with an environment in which they could live, it was possible to cultivate them, but that they could not grow in the ordinary culture media exposed to the air any more than germs like the *bacillus tetani* can.

Veillon has cultivated, isolated, and described several different classes of these bacteria, which appear from his results to play a most important part in the etiology of appendicitis and of peritonitis. In his monograph upon the subject he speaks of the principal ones, under the names of *B. fragilis*, *B. ramosus*, *B. fusiformis*.

In twenty-one of his twenty-two cases Veillon demonstrated the presence of anaerobic germs by means of special methods of cultivation. In two cases they were the only organisms present, while in nineteen cases they were associated with a small number of *streptococci* and *bacillus coli communis*.

He found it necessary to pay the greatest attention to the details of the technique, in order to provide the organisms with the requisite conditions.

The culture medium had to be solid and transparent, and the exclusion of air rigid. The procedure which he followed has been fully described by Dr. Rist (Paris), who has also done much valuable work upon allied subjects, such as the bacteriology of otitic suppurations, where he has arrived at the same conclusions as Veillon as to the importance of anaerobic germs, thereby greatly strengthening the position of Veillon's theory.



Glucose agar is the medium employed. Tubes containing it are heated in a bath to 100° C., in order to melt the agar and to drive off the contained air. At 40° C. the agar is still liquid, but the temperature is not so great as to kill the micro-organisms. The tubes are therefore inoculated at this temperature in the ordinary way with a sterilised pipette, and immediately afterwards are plunged into cold water, so as to minimise the absorption of air during the process of solidification.

In the upper end of the tube there is some admixture with air, and in this layer aerobes grow; while the rest of the column satisfies the conditions required by strict anaerobes. The colonies of anaerobic organisms, therefore, develop in the lower parts of the tube at the end of a variable time, amounting sometimes to eight days. They can easily be seen through the transparent medium. When the colonies are fairly developed they can be separately removed for further examination.

Veillon sums up his conclusions in the following way:—

“All these microbes are strictly anaerobic, and are constantly met in the pus of appendicitis, in abscesses limited to the cæcum, and in peritonitis. Their extreme abundance, shown by examination of stained specimens of the fresh pus and by culture under special conditions, their predominance over the bacillus coli communis and the other aerobes found in these cases, with their exclusive appearance in a few cases, their pathological and chemical properties (gas, fœtor), allow us fairly to affirm that they play an important part in the production of appendicitis and peritonitis. In virtue of their gangrenous properties they are the active agents in producing perforation, and it is to them that the pus owes its fœtor. It is their toxines which give rise to the symptoms of intoxication.”

Rarer forms of peritonitis arise from glanders, actinomycosis and anthrax and various other bacilli, such as bacillus pyocyaneus, pyogenes foetidus typhosus, proteus, lanceolatus, besides the pneumococcus and the gonococcus have been described in connection with it.

Probably, in the production of peritonitis the gonococcus and the pneumococcus only act in combination with the more common pyogenic bacteria. Acute diffuse peritonitis may arise in cases of gonorrhœal salpingitis.

The pathology of rheumatic peritonitis, and of the peritonitis associated with certain forms of renal disease, is obscure, although possibly the two are allied in nature. That rheumatism is a microbic disease is in accordance with the most recent bacteriological work on the subject. The nature of renal disease, and of the peritonitis which arises in connection with it, is much more doubtful.

*Ascitis* (*vide article on Ascitis*).—In this condition the peritoneum is only affected secondarily, and to a slight extent. From venous or lymphatic stasis, fluid exudes into the peritoneal cavity, and in time, from the increased pressure, the membrane becomes opaque and thickened.

Unlike the fluid in serous peritonitis, it shows no power of spontaneous coagulation.

#### NEW GROWTHS OF THE PERITONEUM

*Tubercular Peritonitis*.—The peritoneum is always secondarily affected by tubercle, the infection reaching it from the intestine, from the mesenteric glands, or from the generative organs. In acute cases miliary tubercles of typical form are found scattered over the peritoneum. It is not uncommon also to find a few of them in the peritoneum of patients dying of chronic phthisis. They are studded most thickly over the region of the diaphragmatic peritoneum and the pelvis.



In the more chronic cases the secondary inflammatory changes may be so advanced as to mask the specific changes to a great extent. In these cases the disease shows a striking tendency to simulate tumour. The omentum is rolled up, scarred, and thickened, until it forms a mass which usually lies transversely in the upper part of the abdomen, rather more to the left than the right side. The mesenteries may also be thickened and puckered, causing great deformity.

A certain amount of effusion may be present, but it is not usually very great; occasionally it is hæmorrhagic. Adhesions may lead to the formation of pockets which contain fluid and simulate cysts.

In children especially it is common to find marked affection of the mesenteric glands, which are enlarged and hard with caseation in the centre. It is curious that the association of tubercular peritonitis with tubercular ulceration of the intestine is not more commonly found.

**MALIGNANT DISEASE.**—*Cancer.*—*Primary neoplasm* of the peritoneum is exceedingly rare. Cases of primary epithelioma have, however, been described, although it is probable that they were of the nature of endothelioma rather than true epitheliomata. Endotheliomata occur as nodular growths; they are usually multiple and flattened, the various nodules coalescing to a greater or less extent, and they are white in colour. Sometimes they form large isolated tumours, marrow-like in consistence, the peritoneum round them showing inflammatory thickening. They arise from multiplication of the endothelial cells of the surface of the membrane and of the lymphatics. Microscopically, endotheliomata are characterised by nests or clusters of endothelial cells arranged in a fibrous matrix following the course of the lymphatics.

*Secondary malignant peritonitis* is comparatively common, arising both by extension and metastasis. The disease may start from any of the abdominal organs, but it is most common in connection with cancer of the stomach or ovaries. Discrete nodular masses of growth are found scattered over its surface; there is often much induration and rolling of the omentum, which forms a palpable mass in the hypochondrium as in tubercular peritonitis.

In both primary and secondary carcinoma there is usually some blood-stained exudation in the peritoneal cavity.

*Sarcoma.*—Retroperitoneal sarcoma cannot clinically be distinguished from malignant disease affecting the peritoneum itself, and as there is structural continuity between the peritoneum and the subjacent tissues, it is well not to draw too fine a line of distinction. Sarcoma may originate in the folds of the mesentery and omentum as well as from the retroperitoneal tissue.

Besides sarcoma, various other mesoblastic tumours originate in the retroperitoneal tissues behind the peritoneum, and involve the peritoneum secondarily—ex. lipomata, fibromata, angiomas, besides various kinds of cysts, including hydatids.

Cysts in the mesentery may be multiple and of small size, as in a specimen at the Royal Free Hospital Museum, or they may be single and large.

Inflammation around the foramen of Winslow may obliterate the opening and lead to cystic distension of the lesser sac.

**LITERATURE.**—CLIFFORD ALLBUTT'S *System of Medicine*.—SCHÄFFER'S *Text-Book of Physiology*.—TILLAUX. *Traité d'anatomie*.—TREVES. *Peritonitis*.—*Twentieth Century Practice of Medicine*.—*Bulletins et mémoires de la société médicale des hôpitaux de Paris*, 1899.—*Archives de médecine expérimentale et d'anatomie pathologique*, 1898.



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ETIOLOGY.—This has already been dealt with to a large extent. It is important, however, to mention the actual way in which the bacteria enter the peritoneal cavity.

1. *Wounds.*

(a) Penetrating wounds of the abdominal wall with or without injury to viscera.

(b) Injury to the viscera without any wound of the abdominal wall.

2. *Perforation by a foreign body.*

(a) Those formed in the body—such as gall-stones, enteroliths, etc.

(b) Those taken in by the mouth.

3. *Operations on the abdomen.*

4. *Internal strangulations*, including volvulus, intussusception, and internal herniæ.

5. *Extension from an inflamed organ*, especially intestine or vermiform appendix.

6. *Extension from a local abscess*, of which the most common forms are suppurative appendicitis and suppurative salpingitis.

7. *Perforation of hollow viscera*, as in gastric ulcer, duodenal ulcer, typhoid ulcer, etc.

Of these causes, peritonitis following surgical operations upon the abdomen is becoming much rarer than formerly owing to improvements in operative technique. *By far the most common cause in both sexes is appendicitis.*

*Symptoms.*—When we consider the great variety of causes of acute peritonitis it is not surprising that the symptoms vary exceedingly in different cases. Sometimes we are confronted with a patient whose appearance suggests typical surgical shock; in another case the local evidences of peritonitis are at once obvious; in yet a third the symptoms generally may be so masked that the most experienced clinician may miss the fact that the central feature of the case is peritonitis. There is no definite line of demarcation between those cases which die within twenty-four hours with symptoms of or resembling surgical shock, and those in which the peritoneal symptoms



form so prominent a feature that attention may be withdrawn from the fact that the patient is also suffering from the effects of general septic poisoning.

There is a large number of cases in which the symptoms point to an inflammation of the general peritoneal cavity, but in such, after two or three days, all the general symptoms subside, leaving only the evidence of local mischief, which is usually in the region of the appendix. I have had three opportunities of thoroughly exploring the peritoneal cavity in such cases, having operated under the mistaken diagnosis of a perforated gastric ulcer in two of them, and in the third no diagnosis could be made. In all three cases the symptoms pointed strongly to general peritonitis, but it was found that the peritoneum showed no sign whatever of inflammation. All three cases developed appendicular abscesses within a week. The explanation of this phenomenon will be found later on.

Two kinds of cases may be taken as types of a general peritonitis, viz. that following operations upon the abdomen, and that following an appendicitis where the micro-organisms get through the wall of the appendix without necessarily an actual perforation of the wall of the tube taking place—in other words, there may be a bacterial perforation only. At the present day the former cases are happily becoming more and more uncommon, and therefore it will be better for us to take for our description a case where the trouble has started in the appendix without a sudden perforation of that organ, a condition which is the most common cause of general peritonitis. There are the very acute cases before referred to, in which the patient appears to be suffering from profound shock and septic poisoning. In a third group of cases the symptoms of peritonitis are slight compared with the mischief that is found at the operation or autopsy; these cases are termed *latent* or *masked* peritonitis. In dealing with a typical case of acute peritonitis it will be best to describe fairly briefly the symptoms and course of the disease, and then to consider each important symptom in detail.

A TYPICAL CASE OF ACUTE PERITONITIS.—The patient begins to feel unwell, and within an hour or two he complains of cold, and may even shiver; not uncommonly there may be an actual rigor ushering in the symptoms. In nearly all cases the first definite symptom that makes its appearance is *pain* which, though very constant in its presence, is very variable in its character and in its intensity. When the pain is slight it generally has a curious tendency to disturb and worry the patient out of all proportion to its intensity, and this confers upon the face a somewhat characteristic expression of anxiety. On the other hand, the actual intensity of the pain may be so great that it is quite alarming both to the patient and his friends. At first the pain is referred as a rule to the whole of the abdomen, but later on it is generally localised to the region of the umbilicus. As a rule the *abdomen* is rigid, and the *skin* of the whole or of a part of the abdomen is markedly hyperæsthetic. In order to diminish the abdominal tension as much as possible the patient lies on his back with the thighs flexed and the shoulders raised. When the costal respiration is much hampered the arms are often raised above the head, so as to bring into action the extraordinary muscles of respiration. Owing to this rigidity of the abdominal muscles and to the abdominal distension the *respirations* become more and more costal in type. The *pulse* is increased in frequency, is of high tension and small; this high tension is characteristic, and seems to be one of the early effects of the septic poisoning upon the vasomotor system. This small yet high tension pulse is so characteristic that it has



gained for itself the term *wiry*. This must not be confused with the pulse of a later stage, which is often termed *thready*, owing to its low tension and small volume. The *temperature* rises rather rapidly to  $103^{\circ}$  or  $104^{\circ}$ , and after this initial rise it generally falls a little, maintaining an average of about  $101.5^{\circ}$ . After the first 24 or 36 hours the hard and somewhat retracted abdomen begins to distend, and the percussion note becomes more and more resonant and finally *tympanitic*. The distension rapidly increases, so that the abdomen becomes blown out and tense. *Vomiting*, which usually sets in about the same time as the pain, is a prominent feature of the disease. At first the ordinary stomach contents are brought up, when the vomit is yellow, being distinctly bile-stained, and this is followed by a characteristic green liquid, which is finally succeeded by a brownish black one having an offensive odour distinctly faecal in character. At the commencement of the inflammation the *bowels* may be loose, but as soon as the abdomen distends constipation sets in. The act of *micturition* is often frequent at first, and later on there is usually retention, especially if the origin of the trouble is in the appendix or in the pelvic organs. The urine is small in quantity and high coloured, usually containing abundance of indican. There is usually free fluid, causing dulness in the flanks, which may shift on rotating the patient.

When all the symptoms are fully developed the aspect of the patient becomes very characteristic; so well was it described by Hippocrates, that it is often termed the "*facies hippocratica*," viz. "sharp nose, sunken eyeballs and hollow temples, the ears cold and pinched, with their lobules turned outwards; the skin of the forehead is rough, puffy, and dry; the colour of the whole face being brown black, livid, or leaden"; this excellent description requires modification in the last statement only, where we must remember that the face described belonged in all probability to the Oriental type.

This typical case of acute peritonitis usually lasts from five to ten days, nearly all the symptoms increasing in severity until the fatal end, which is usually heralded by the skin of the face and hands becoming cold and clammy, and tinged with a dark blue colour.

SOME OF THE MORE IMPORTANT SYMPTOMS IN DETAIL.—*The onset*.—In most cases the onset is sudden, but this largely depends upon the actual way in which the invasion of the general peritoneal cavity takes place. If it is due to the perforation of one of the hollow viscera, such as the stomach or intestine, without any previous inflammation of the peritoneum which might tend to localise the escaping contents, the onset comes with alarming suddenness. Literally within a minute the patient may pass from a condition of apparent health to a condition of collapse and shock, that has been known to end fatally within an hour. In other cases, where an organ, generally the appendix, has become inflamed for some time, the invasion of the general peritoneal cavity is comparatively speaking gradual, and it may take from twelve to twenty-four hours before it can be said that acute general peritonitis is definitely established. When very acute the onset is associated with a rapidly falling temperature. In other cases there may be a rigor and a temperature of  $104^{\circ}$  or  $105^{\circ}$  within an hour or two of the commencement of the attack.

*Shock and "Peritonism."*—The shock produced by an extensive scald of the skin, or by the skin being badly lacerated, is extreme, and bears a very definite proportion to the area of skin involved. In the same way any extensive lesion of the peritoneum produces a similar condition,



whether it be due to the irritation of the contents of the stomach, as for example in perforation of a gastric ulcer, or to the torsion of an ovarian pedicle.

*Pain.*—This is nearly always the earliest symptom, and may vary from one that is accurately described by the term *agonising*, to one that is slight in intensity, but of the character described above (see p. 306). At first the pain is generalised over the whole abdomen, but later on it is usually referred by the patient to some definite area; very often the region of the umbilicus seems to be the most painful. In cases where an inflammation of a definite organ has given rise to the peritonitis the pain may be referred to the region of that organ, especially in the case of the appendix.

*Tenderness.*—This tenderness may be of two kinds:—

1. Superficial tenderness or hyperæsthesia of the skin.
2. Deep tenderness, which certainly is present in the parietal peritoneum, but may also be present in the muscles or the underlying viscera. The tenderness of inflamed parietal peritoneum is very marked, and is often verified when laparotomy is performed under local anæsthesia. The great difficulty of proceeding with the operation is the exquisite tenderness of the parietal layer of the peritoneum (see p. 312). When the superficial tenderness is very extensive it is probably associated with inflammation of the intestines, especially the small gut. According to Head, the superficial tenderness is a referred tenderness from one or more of the organs of the abdomen, and the tenderness would follow the rules laid down by him. For instance, in the case of the intestines being inflamed the superficial tenderness would not necessarily be limited to the front of the abdomen, but there might be a bed of hyperæsthesia running to the back or downwards towards the groin. On the other hand, the deep tenderness is due to the direct pressure upon the peritoneum, and is obtainable at any spot where the inflamed peritoneum can be pressed upon. In general peritonitis this deep tenderness, therefore, can be elicited practically over the whole of the abdomen, with the exception of the area posterior to a line drawn from the anterior superior iliac spine to the tip of the last rib, where it is not easy to press directly upon the peritoneum. In the early stages of general peritonitis the superficial tenderness is nearly always extremely well marked, but as the inflammation progresses it begins to diminish, and may finally disappear, whereas the deep tenderness progressively increases until the general peritonitis is well developed. In nearly all cases of general peritonitis some organ of the abdomen is first affected, and this organ may continue to present its referred superficial tenderness after the general peritonitis has become established. In several cases of general peritonitis arising from appendicitis the writer has found an extensive area of superficial tenderness below the level of the umbilicus co-existing with the deep tenderness, and at the operation advanced general peritonitis has been found. On this point, therefore, we may make two positive assertions, namely, that if the tenderness is only superficial there is no marked affection of the peritoneum, and that in cases of undoubted suppurative peritonitis due to appendicitis, and probably in any other of the ordinary causes, there may be superficial tenderness existing at the same time as the deep.

Sometimes, in an attack of appendicitis in which the inflammation throughout remains localised, the superficial tenderness and rigidity of the abdominal wall may involve the whole of the abdomen in the early stages of the disease. In such cases the symptoms are highly suggestive of the



onset of general peritonitis, and when associated with marked signs of collapse the diagnosis of a ruptured ulcer is sometimes almost forced upon one by the serious condition of the patient. This generalisation of the abdominal signs are probably of reflex origin, the inflammation in the region of the appendix forming a spintus strong enough to cause a spreading in the central nervous system from the representation of the appendix to that of the whole of the abdominal viscera. It is this class of case that is referred to in the first paragraph of p. 306. In two of these cases the collapse was so great that the diagnosis of perforated gastric ulcer seemed inevitable; yet at the operation the general peritoneal cavity presented no sign whatever of early inflammation.

Another explanation that occurs to one is that at first there is a bacterial invasion of the general peritoneal cavity, but that the peritoneum has sufficient resisting power to repel the invasion except at the spot where the bacterial activity is most intense, *i.e.* in and around the appendix. If, however, the view now generally held, that inflammation is the necessary defensive and offensive weapon against bacterial assaults be true, there should be evidences obtained at the exploratory operation that such an attempt on the part of the peritoneum has been made. In the two cases already mentioned there was not the slightest trace of any inflammatory reaction in the peritoneal cavity, and therefore I think we may safely conclude that the generalisation of the signs is due to a widespread disturbance in the central nervous system.

*Vomiting.*—Next to pain this is the earliest, most constant, most distressing, and most persistent symptom. The abdominal muscles take little if any part in the expulsive act, so that the stomach itself does most of the work; hence the act of vomiting appears to occur without much effort. As to the causation of the vomiting it is very difficult to make anything like an accurate statement. In the early stages it is probably due to a large extent to the poisoning of the centre in the medulla oblongata, or to some reflex nervous action from the inflamed peritoneum, the former being the more probable. Of course, after paralysis of the intestine has set in we have another factor at play very much resembling the condition of affairs in intestinal obstruction. Like most of the symptoms of peritonitis vomiting is very variable, and may be slight or even absent. This is especially the case in the variety known as *masked* peritonitis.

*Defæcation.*—In the early hours of the attack there is very often a stool which is generally described as being loose; diarrhœa is not uncommon. A frequent desire to defæcate without any result is sometimes met with, especially when the inflammation has its origin in the appendix or pelvic organs, and this may be so intense as to deserve the name *tenesmus*. As soon as the distension of the small gut sets in the colon becomes inert, and constipation is the rule in fully developed peritonitis.

*Distension of the Abdomen (Meteorism).*—As the intestinal coils become paralysed and distended, and the abdominal muscles begin to relax, we get the condition of *meteorism*, which gradually increases until the abdomen is much distended. This gradual distension of the abdomen is undoubtedly diagnostic of acute general peritonitis, and in the opinion of the writer it is a certain indication that no delay should occur before opening the abdomen. Of all the symptoms of peritonitis distension of the abdomen is perhaps the most constant, and however much one may waver in the early stages of the disease, this physical sign should disperse all trace of hesitation concerning



the nature of the trouble. As a rule it sets in at the end of the first forty-eight hours, but in the less acute cases it may not set in for three or even four days. There seems no doubt that the early stage of meteorism is due to distension of the small intestine, and only quite later in the course of the disease does the large gut become much distended. In my experience it is seldom that the distension of the large gut gives any trouble at an abdominal section for acute peritonitis, whereas in most cases the distended coils of small gut are most troublesome to the operator.

*Temperature.*—This is the most variable of the whole of the symptoms, so variable, indeed, that it is safer for the practitioner to place no importance whatever upon it so far as the diagnosis and treatment of the disease are concerned. As a rule, in most diseases great stress is laid upon the importance of the temperature chart in diagnosis, and in my experience there is too great a tendency to lay stress upon, for instance, the absence of fever in a suspected case of peritonitis as throwing doubt upon the nature of the disease. It is better, therefore, to underrate rather than overrate the value of this symptom. In cases of sudden perforation of the viscera the temperature is usually subnormal, and often remains so until the end.

*METHOD OF EXAMINATION.*—The patient should be placed on his back with the legs extended and the arms by the side of the body. The body is uncovered to such an extent as to show the apex beat of the heart above and Poupart's ligament below. As the examination proceeds as much of the body should be covered up as is possible. By *inspection* attention should be paid to the movements of the abdominal muscles in relation to respiration, especially whether a part or the whole of the muscles are acting or not. Next, it should be noted whether the abdomen is retracted or distended. In the later cases any discoloration of the surface should be noticed, especially any discoloration around the superficial veins. By *palpation* we should carefully test the superficial tenderness and the deep tenderness. In examining the abdomen with the hand it is advisable to commence on the skin of the thorax, so that the apprehensions of the patient are minimised. By *percussion* we may learn something from the resonance of the note whether uniform or not, and when the resonance is suggestive of *tympanites*. If the liver and splenic dulness are practically obliterated the diagnosis of free gas in the peritoneal cavity is probable. Very little aid is gained by *auscultation*; it is interesting to note, however, that the great physician Bright was the first to point out the existence of the friction sound in early stages of peritonitis, especially in the chronic forms. It is most important that the patient should always be examined *per rectum* and *per vaginam* to ascertain the condition of the pelvic organs.

#### CLINICAL VARIETIES

*Peritonitis fulminans, Ultra-acute Peritonitis.*—All these cases are usually caused by the perforation of some part of the alimentary canal in which no adhesions have formed from previous inflammation. The patient is suddenly seized with most acute pain, and within a few minutes is suffering from the most profound shock. As the bacterial invasion proceeds symptoms of septic poisoning rapidly develop, and the patient dies within twenty-four to forty-eight hours, without having rallied sufficiently for operative interference to be seriously entertained.



There is a group of cases following operations upon the abdomen, now happily becoming less and less common, in which the patient never really recovers from the shock of the operation, but dies within twenty-four to forty-eight hours with a rather distended abdomen. At the post-mortem examination there is scarcely any evidence of inflammation of the peritoneum, certainly not enough to justify us in saying that the patient dies of peritonitis. Most surgeons consider that they are justified in looking upon this result as death from shock, and consider that the intestinal distension is due to some special reflex nervous mechanism. Some pathologists, on the other hand, have found the peritoneal cavity to be infected with micro-organisms, and they look upon these cases as instances of a very rapid invasion of the peritoneum by virulent bacteria, which grow so rapidly that inflammation has no time to develop. A definite series of accurate bacteriological investigations alone can settle this point.

*Latent or Masked Peritonitis.*—The patient walks into the hospital and does not feel particularly ill; he usually states that his illness commenced four or five days previously, but that until that particular day he was not ill enough to seek advice. Within two or three days he suddenly becomes collapsed, and dies, with his peritoneal cavity full of pus. Owing to the absence of urgent symptoms the surgeon's help is often not summoned until the patient is too collapsed for operative interference to be anything more than a forlorn hope.

*Peritonitis of the New-born Child.*—These cases are due to one of two causes: (1) The septic infection of the umbilical cord, due probably to faulty technique on the part of the accoucheur or midwife; (2) Rupture of the distended large intestine, which may be due to the pressure of parturition, but more likely to be the giving way of some small ulcer in the colon.

*Peritonitis caused by Cutaneous Erysipelas.*—In rare instances erysipelas of the abdominal wall has been followed by fatal peritonitis. This is difficult to explain, because the lymphatics of the abdominal wall do not communicate with those of the peritoneum. The symptoms of the peritonitis are often masked, and not even suspected during life.

*Peritonitis in the Course of Acute Specific Fevers.*—Peritonitis is most commonly met with in scarlet fever, as we might expect from the tendency of this disease to develop dangerous complications. In typhoid fever it may occur as the direct result of a perforating ulcer, or be due to a bacterial penetration of the wall of the alimentary canal without any physical breach of continuity. Peritonitis has also been met with in the course of measles, smallpox, and typhus fever.

*Rheumatic Peritonitis.*—Although doubt has often been thrown upon the existence of this complication of rheumatic fever, it is definitely proved that acute inflammation of the peritoneum does sometimes occur as a complication of acute rheumatic fever. It usually terminates favourably after a few days, although fatal cases have been reported.

*Peritonitis in Bright's Disease.*—This is a rare complication. The mode of origin of the peritonitis in this disease, as in the case of acute rheumatic fever, is possibly as follows:—A purely chemical peritonitis is set up by toxins. After a time the wall of the intestine is sufficiently injured by this chemical peritonitis to allow the organisms contained in the intestinal contents to work their way through to the peritoneal cavity.

*Idiopathic or "Cryptogenetic" Peritonitis.*—The more carefully cases



are examined by improved modern methods the rarer does the diagnosis of idiopathic peritonitis become. At present there are two groups of cases of which the origin is absolutely obscure, and hence it is convenient to place them under this heading. In the one group the patients are children, chiefly girls, and in the other the victims are adults. In both these cases a chill is generally quoted as being the cause. The attacks of enteritis with diarrhoea so commonly met with in Europeans living in hot countries, especially in Egypt, seem to be due solely to some reflex vasomotor disturbance from the sudden chilling of the body in an atmosphere nine times drier than the one they have been accustomed to. The cooling of the body by means of perspiration is therefore rapid, the atmosphere being very hygroscopic, so that a reflex effect is produced, causing a dilatation of the vessels of the splanchnic area. If this condition is neglected it is easy to see how some inflamed area of the gut may prove a weak spot, and allow the germs to penetrate to the peritoneal cavity.

DIAGNOSIS.—Quite at the onset it may be very difficult indeed to diagnose the existence of peritonitis. Severe abdominal pain may be caused by several conditions other than peritonitis, *e.g.* in acute intestinal catarrh (entero-colitis), in which diarrhoea and intermittent colic are the chief symptoms. The diagnosis from intestinal obstruction may be quite impossible for the first two days, and sometimes throughout the disease. This point is not of much importance, as in either case the surgeon would operate without delay. Sometimes the onset of an acute pleuro-pneumonia may give rise to referred tenderness in the abdomen highly suggestive of commencing peritonitis. There is a condition known as *hysterical* peritonitis met with on the Continent more frequently than in this country. There are certain rare conditions, such as *acute hæmorrhagic pancreatitis*, or a *ruptured tubal pregnancy*. One may admit at once that the importance of accurate diagnosis in acute inflammation of the abdomen is of minor importance. In almost all cases the surgeon has very little doubt that the condition is one for which an exploratory incision is definitely called for. In the early stage of any disease resembling peritonitis an exploratory incision would do the patient but little harm, and if the surgeon waits until his diagnosis is very certain the hopes of saving the patient become enormously lessened. The difficult question that so often arises, namely, whether the inflammation is general or local, matters but little at the present day; in both conditions early operation is indicated.

TREATMENT.—*Indications for Operation.*—With the few exceptions that make the rule it is admitted on all sides that if an acute general peritonitis becomes once established, and is allowed to run its course without surgical interference, the termination is always a fatal one. The question of the indications for operation in a case of acute general peritonitis has passed through a rapid series of changes during the past ten years. At first the hesitation in diagnosis between general and local peritonitis was allowed to postpone an operation until either it was too late to save the patient if the case developed into general peritonitis, or it resolved itself simply into a question of opening a localised abscess at the end of a week or ten days. Owing to the extreme difficulty in making sure whether the trouble is going to remain localised or not, surgeons are beginning more and more to agree that operative interference should be undertaken as soon as the diagnosis of peritonitis is made, whatever it is thought might be the extent of the inflammation. I can see no way out of the difficulty, excepting to act on a uniform plan, and always to operate as soon as the diagnosis of peritonitis is made, providing the general condition of the patient justifies the procedure.



It is extremely difficult to gauge the resisting power of a patient with acute general peritonitis. Sometimes a case apparently hopeless in every respect, with signs of extreme poisoning, with abundance of pus, and with distended gut, recovers rapidly after a judicious laparotomy. On the other hand, a patient with excellent pulse, and no grave signs that can be gauged, will die a short time after the operation. Of course it is most important to ascertain what drugs the patient has been having since the commencement of the illness—opium and morphine establish a false standard of strength; but this important question will be dealt with at some length later on. Successful results in the operative treatment of this disease depend to an enormous extent upon the judgment of the operator, who has to consider, first of all, whether a thorough attempt can be made to deal with the case with a general anæsthetic; in which event laparotomy, flushing, and drainage of the peritoneal cavity must be promptly carried out. But if he is of opinion that the patient cannot stand this ordeal, it may be possible to anæsthetise the patient for a short time, so that an abdominal section can be rapidly performed, and a drainage tube with gauze packing introduced. In very bad cases, where a general anæsthetic is obviously quite out of the question, the surgeon must then attempt to do what is necessary with a local anæsthetic. In my experience these cases do far better than one anticipates.

Even with acute lobar pneumonia I have found it possible to remove a suppurating ovary in a case of acute general peritonitis, with recovery of the patient. Indeed, whenever there is marked pulmonary infection in cases of acute general peritonitis it is eminently desirable to attempt at any rate to deal with the case with local anæsthesia. If the patient cannot stand the pain chloroform can then be given. It is the parietal peritoneum that is so very tender, and I have found that a  $2\frac{1}{2}$  per cent solution of cocaine very rapidly effects anæsthesia over a large area. The details of these operative procedures may be described under the following three headings:—

1. Laparotomy, flushing, and drainage.
2. Rapid laparotomy with drainage.
3. Laparotomy with local anæsthesia.

1. *Laparotomy, Flushing, and Drainage.*—The patient being under the influence of chloroform a free incision is made in the middle line below the umbilicus. There is a considerable diversity of opinion amongst surgeons concerning the best anæsthetic for abdominal work. From a surgeon's point of view alone it seems to the writer that chloroform is the best, both for the rapid and comfortable performance of the operation and for the absence of the pulmonary disturbances so common with ether. From the anæsthetist's point of view, which is concerned solely with the patient's safety during the operation, the consensus of opinion in London is strongly in favour of ether. With a competent chloroformist the writer considers that the safety of the patient during the operation and the few succeeding days is greater with chloroform than with ether. The abdomen being opened, the surgeon soon finds whether the case is a general one or not. A search is then made for the seat of origin of the trouble, the right iliac fossa being the starting-point. If the appendix is the offending organ, an incision should be made at a spot over it most convenient for its removal, and if possible the appendix is excised. Sometimes it is possible to remove the appendix through the median incision, but as a rule it is much more satisfactorily dealt with by two incisions. All traces of local suppuration should be flushed out, and the region carefully sponged. The part of the abdomen in the region of



the appendix, especially the cavity of the pelvis, should be well flushed out with a normal solution of the temperature of the body. An indiarubber drainage tube is placed in contact with the spot where the disease originated, whether appendix, Fallopian tubes, or elsewhere, and round this tube some iodoform gauze should be packed. The median wound should then be closed, and one or two stitches placed in the lateral incision so as to prevent prolapse of intestine, and at the same time not to interfere with effective drainage. If no local focus can be found the abdominal wound may be closed, after thoroughly flushing the peritoneal cavity. This is a convenient place for discussing the question of drainage of the peritoneum.

*Technique of Drainage of the Peritoneum.*—If a tube be inserted, say six inches from the skin down into the iliac fossa, and carefully examined at an autopsy twenty-four hours afterwards, it will be seen that the tube is surrounded by coils of intestine, which are sealed together with exudation which completely prevents the tube receiving any appreciative amount of the general peritoneal fluid. The surface of the coils surrounding the tube exudes the turbid serum, which fills the test-tube-like cavity. As soon as this liquid is removed by the syringe or by gauze the coils gradually exude another quantity. Unless the tube reaches directly down to a septic focus it seems to be of little use in draining the general peritoneal cavity. By leading down to a septic focus it helps to form a well-protected channel for the exit of any discharge from the suppurating tissue. On the whole, it seems that drainage tubes, whether made of glass or indiarubber, are quite useless as agents for draining the general cavity of the peritoneum, even when present in numbers, and indeed may do harm by mechanically irritating the coils of gut. We may sum up the rôle played by the drainage tube by stating that its function is to obtain a channel through the coils of intestine by which some septic focus can discharge its contents to the exterior with ease. There is one exception to this rule, and that is in the case of the pelvis. Here there is naturally a tendency for fluid to collect in a definite quantity, so that a tube reaching to the bottom of the pelvis must assist in draining that basin. The question of draining the pelvis is a very important one, and some surgeons prefer to drain Douglas's pouch in women by an incision through the vagina,—a proceeding, however, which necessitates additional shock, so important a factor in cases of acute peritonitis, and of which the necessity is doubtful if efficient drainage is secured by a tube and gauze from above. Several modes of draining the peritoneal cavity efficiently have been attempted, but not one has met with even moderate acceptance. Numerous strands of gauze, like a cat-o'-nine-tails, have been used, but I think with more damage to the intestine than good to the patient; incisions through the skin in the loins to drain the region of the colon and kidney, a region where pus does not collect to any marked extent, and many others equally futile. On the whole, therefore, we must trust to efficient removal of the fluid, whether serum or pus, by flushing and by making sure that the focus of origin is effectively removed or placed in connection with the exterior by means of suitable drainage.

One of the most disappointing features in the surgical treatment of general peritonitis is the tendency to distension of the intestine, especially of the small gut. Sometimes, after a thorough laparotomy, flushing, and drainage of the peritoneal cavity without markedly depressing the patient, there is the difficult complication of distension of the intestine to be overcome. The reasons for this distension have been already given, and it is chiefly to the late Greig Smith of Bristol that we are indebted for pointing



out how very important it is to drain the contents of the alimentary tube as well as the peritoneal cavity. The difficulty, however, lies in the fact that when the intestine is paralysed it is quite as difficult to effectively drain a long extent of tube as it is to drain a large extent of peritoneal cavity. Over and over again one is disappointed in the results of opening distended coils of small gut. The particular coil, say eighteen to twenty-four inches, rapidly collapses, but beyond that it is extremely difficult in many cases to get rid of much more distension. This difficulty is in marked contrast with the relief of distension in intestinal obstruction,—those cases of intestinal obstruction in which there is no marked peritonitis. If the intestines are much distended one of the longest coils should be incised, and a T-shaped glass tube inserted, with a thin collapsible indiarubber tube attached to it. Very often, after a few hours, the distended coils will begin to contract and expel the contents, although one may be very disappointed with the result at the time of operation. In these desperate cases the first point is to save the patient's life; if recovery ensues surgery can readily deal with the artificial anus that is formed.

2. *Rapid Laparotomy with Drainage.*—The patient is quickly anæsthetised, and as soon as the abdominal wall is flaccid a median incision is made, and any inflammatory exudation is allowed to escape; if rapid examination reveals the focus of origin a tube and gauze drain are inserted down to the suppurating spot. If the seat of origin cannot be found promptly a gauze drain may be left in the peritoneal cavity and the wound partially closed. If the intestine is much dilated the first distended coil should be opened, and a T-shaped glass tube should be inserted as already described.

3. *Laparotomy with Local Anæsthesia.*—The skin of the abdomen in the middle line should be rendered anæsthetic with  $\beta$ -eucaine. As much should be done as the patient can bear under the conditions of local anæsthesia.

In no department of surgery is it more important for the surgeon to form his opinion with decision, to commence operation without delay, and to operate quickly. *Nothing is more certain than that every additional minute of exposure in the case of an abdominal section diminishes the patient's chances of recovery.*

AFTER-TREATMENT.—1. There is nearly always a great deal of *restlessness* after laparotomy for general peritonitis, and it is generally necessary to give a hypodermic injection of morphine (gr.  $\frac{1}{4}$ ). Very often the patients are much collapsed, and a hypodermic injection of strychnine (gr.  $\frac{1}{30}$  to gr.  $\frac{1}{15}$ ) acts with an immediately good result. If the pulse becomes very feeble, it is advisable to “infuse” the patient with a normal saline solution, *taking care that the solution is maintained at the body temperature as it enters.* Nothing depresses the patient more than to infuse a pint or two of solution at a temperature less than that of the body. In bad cases this “infusion” is best given through the basilic vein. In moderate cases of collapse half-pints of hot water placed into the rectum at frequent intervals are rapidly absorbed.

2. It is most important to keep the alimentary canal at rest, and therefore the introduction of *food* into the stomach should be prohibited. All food should be given by the rectum. Perhaps the best is peptonised beef-tea and small quantities of brandy. So far as nourishment is concerned, the patient obtains quite sufficient to tide him over the crisis of the disease. As a rule, if a patient is alive and improving on the fourth day after the operation he will recover.

3. One of the most constant and difficult symptoms to treat is *thirst*.



The general sensation of thirst is best satisfied by injections of warm water per rectum, but the local condition of thirst, where the patient craves for something by the mouth, a little warm water, or if much preferred, a little iced water, may be given. In cases of excessive thirst a feeder full of water (5 oz.) may be given; even if this is vomited no great harm is done, provided the act is not frequently repeated.

4. The toilet of the *tongue* and *mouth* generally is most important in these cases. A little care on the part of the nurse in scraping away the débris from the furred tongue and from the dry lips and mucous membrane of the cheeks relieves a great deal of the patient's suffering. A solution of bicarbonate of soda (20 grs. to the oz.) is a good solution for removing the decomposing material, and afterwards a little weak carbolic lotion (1 in 80) is a good mouth disinfectant.

5. After the operation no *aperient* should be given for the first twenty-four hours. As soon as the patient has rallied sufficiently from the operation, and if there is distension of the intestine, an enema of water (a pint) combined with tincture of valerian (1 drachm) should be given. If at the end of twenty-four hours the patient is improving, small doses of sulphate of magnesium may be given, say ʒj. of each, every three hours, or small doses of calomel (gr.  $\frac{1}{2}$ ) every three hours.

THE ADMINISTRATION OF OPIUM AND MORPHINE.—Before the question of operative treatment has been discussed and definitely decided upon, no opium or morphine in any form should be given. The administration of this drug causes an immediate improvement in all the symptoms; the pain is diminished, the rigidity of the abdominal muscles may disappear altogether, and the patient's general condition is so much improved that the surgeon may be quite deceived as to the gravity of the case. Many lives have been lost owing to the false appearance of improvement brought about by the administration of this drug. The patient's improvement may be maintained until suddenly the dose of poison absorbed from the peritoneal surface reaches its maximum, and the patient suddenly collapses. If the case is considered too bad for operative interference, then morphia may be given until the patient is quite relieved of all distressing symptoms. After the operation, we have already alluded to the value of morphine in quieting the nervous system and also in stimulating the action of the heart. In all post-operation cases, however, morphine should be given with especial care that no inertia of the intestines is produced.

NON-OPERATIVE TREATMENT.—According to our present knowledge there is no reasonable method of attempting to cure a case of acute general peritonitis by medicinal means. One might be inclined to take exception to this strong statement in the case of general peritonitis without any local lesion. For instance, it is possible that a case of gonorrhœal peritonitis, where the infection has travelled up the Fallopian tubes, might recover without operative interference. Such cases, however, are very rare, and it would be impossible to recognise them before opening the abdomen. Even in cases too much collapsed for surgical interference the treatment must be directed to the alleviation of all distressing symptoms.

The method of treatment which has gained considerable ground on the Continent, especially in France, is the subcutaneous and intravenous injection of saline solution. The principle is to introduce into the vascular system, directly by the veins or indirectly by the subcutaneous lymphatics, a saline solution with a view to washing out the lymphatic vessels and blood-vessels, and so enabling the liver and kidneys to get rid of the toxins. This



method, combined with operative interference, is undoubtedly an excellent combination, and seems perfectly rational. Indeed, many of my patients have the "infusion" of several pints of saline fluid if at all collapsed after the operation. Some French surgeons, however, look upon the treatment as sufficient in itself—*e.g.* Michaux says, "Qu'on ne sauvera pas tous les malades, mais qu'on aura certainement la joie d'arracher à la mort quelques-uns de ceux dont l'état semblait le plus désespéré." The solution generally used is sodium chloride 5 grams, sodium sulphate 10 grams, distilled water 1 litre. This is injected at the temperature of the body either into the subcutaneous tissue, or through the median basilic vein (see "Saline Solution, Infusion of"). According to French surgeons, remarkable improvement and cures have been attained.

**PERITONITIS, *Acute Localised*.**—Acute inflammation of a portion of the peritoneum, due to some lesion of the organ it is covering, is a very common occurrence. It may occur in the peritoneum covering the liver, and spread thence to the peritoneum covering the lower surface of the diaphragm. It not unfrequently occurs in the peritoneum covering the stomach in the neighbourhood of a gastric ulcer. Of all the abdominal organs, the vermiform appendix is the one most commonly affected. In women there is frequently local peritonitis round the uterus and Fallopian tubes, usually termed pelvic peritonitis. The chief varieties we will consider at greater length.

1. *Sub-phrenic Abscess*.—Inflammatory conditions may spread from the pleural cavities through the diaphragm to the abdomen. As a rule, however, sub-phrenic peritonitis spreads from the abdominal organs, chiefly the liver and stomach, often developing into an abscess. When the inflammation spreads from the liver, and suppuration occurs, the abscess forms between the right half of the diaphragm and the upper surface of the liver, and is very often diagnosed as an empyema. On the other hand, the sub-phrenic abscess caused by lesions of the stomach forms on the left side. Perforations of certain parts of the stomach or the duodenum may cause effusion into the lesser sac of the peritoneum, and, according to Jordan Lloyd, pathological distension of the lesser peritoneal sac gives rise to a tumour in the left hypochondriac, epigastric, and umbilical regions of a somewhat characteristic shape, but which appears to vary from time to time in form and size according to the condition of the overlying stomach; for when the viscus is full of liquid contents it increases the area of the tumour's dullness, while it makes its outlines less definable to palpation, and if the stomach is distended with gas the dull area becomes resonant, and apparently the tumour may disappear altogether. The colon always lies below the tumour, and never in front of or above it, as is the case in kidney enlargement.

There is a curious form of sub-phrenic abscess which contains gas, and it may very closely simulate pneumothorax. As a rule it is caused by the perforation of a gastric ulcer; less frequent causes are the perforation of a duodenal ulcer; of appendicitis; and of the perforation of abscesses in connection with the kidney, liver, spleen, etc.

*Symptoms*.—These naturally vary a good deal as the primary causes are so diverse. The onset is, as a rule, abrupt, especially when due to a perforation of a gastric ulcer. Besides the severe pain and vomiting, respiration is markedly embarrassed, probably owing to the involvement of the diaphragm. In addition to these symptoms there are the ordinary ones associated with suppuration in a confined space in the abdomen.



Later on abscesses may spread through the diaphragm into the pleura or lung, and the pus may be coughed up by the patient.

*Diagnosis.*—This seems to be very difficult if we may judge from the mistakes that are constantly made by physicians in asking surgeons to open a collection of pus in the region of the diaphragm. When the abscess is between the liver and the diaphragm it is nearly always mistaken for an empyema. When the abscess cavity contains gas, and is situated on the right side, the diaphragm may be pushed up to the level of the second or third rib, and we may get the physical signs of pneumothorax, the liver being usually greatly depressed. When the gas-containing abscess is in the lesser sac of the peritoneum it tends to give the signs of pneumothorax on the left side.

*Treatment.*—As soon as pus is suspected in the region of the diaphragm an exploring needle should be used. If the result is positive some indication is obtained as to the situation of the pus and its depth from the surface. If the needle gives a negative result the surgeon must proceed to make a careful dissection down to the diaphragm in the region of its attachment to the ribs. Very often the pleural cavity overlaps the displaced diaphragm to the extent of several inches. In such cases, if the condition of the patient will allow, the lower end of the parietal pleura may be stitched to the chest-wall, and the wound plugged with iodoform gauze for twenty-four to thirty-six hours, until adhesion has formed to a sufficient extent to allow the surgeon to cut straight through the diaphragm. If after removing a piece of rib it is seen that the pleural cavity is normal, the surgeon should extend the section in a downward direction until the point of reflection of the pleura is seen. The pleura may then be pushed up, and gauze packed tightly in the upper part of the wound. If immediate evacuation of pus is necessary the diaphragm may be incised, the pus quickly removed, and the edges of the incised diaphragm stitched to the edges of the wound. The risk of infecting the pleural cavity is by no means a slight one; it is not due so much to the pus escaping into the pleural cavity at the time of the operation as to the soaking of pus into the surrounding tissues within the few hours succeeding the operation, especially if drainage from the abscess cavity is not quite free, or if the upper part of the wound, in the region of the pleura, is not carefully and thoroughly packed with iodoform gauze. There is one consolation for the surgeon, viz. secondary infection of the pleura is common in subphrenic abscess, even before operation, so that if the germs have reached the pleura before the surgeon, nothing the latter can do is likely to prevent the development of an empyema. The following rules may be worth remembering in dealing with the surgery of subphrenic abscess: (1) When *pus has been discovered* by the exploring needle at a certain spot an incision should be made with that spot as its centre; (2) If *no pus* has been obtained by the needle make a careful exploratory operation through a four-inch incision over the tenth rib in the mid-axillary line; (3) If *no great urgency*, pack the exposed pleura or pleural cavity with sterilised gauze, and wait twenty-four to thirty-six hours before incising the diaphragm; (4) If evacuation of the pus *is urgent*, try and expose the lower reflection of pleura, push it upwards from the ribs and pack tightly with gauze, incise diaphragm, and stitch it to the skin, insuring free drainage.

2. *Abscess around Appendix.*—Owing to the inconstant position of the appendix the situation of the abscess is, of course, variable. Usually it is in the right iliac fossa, with the cæcum on its outer side; sometimes it is wholly within the pelvis; it may be to the left side of the median line, in



the iliac region. An important situation is *submesenteric*, the abscess being limited above by the oblique line of the mesenteric attachment—viz., from the right sacro-iliac synchondrosis to the left side of the second lumbar vertebra—below it reaches the hollow of the sacrum, being variably limited by coils of the small gut; to the right side is the cæcum; in front the mesentery itself and numerous coils of gut form the boundary, so that the general peritoneal cavity is exposed after cutting through the abdominal wall.

TREATMENT.—1. *The usual Type of Abscess*.—When there is a dull area in the iliac fossa the incision should be made over the spot of maximum dullness. The cutting should be done carefully, so that any weak adhesions may be left in order to keep the pus from spreading. *In all cases the whole of the pus should be removed at once and the cavity thoroughly washed out.* If the appendix can be easily got at it should be excised; as a rule, however, it forms a part of the abscess wall and can neither be felt nor seen. There is often a faecal concretion present, which usually escapes through the gangrenous aperture in the appendix, and it is important to try and get hold of this. If it does not come out with thorough flushing, I have found that a blunt Volkmann's spoon, very gently used, is generally efficient. A good-sized drainage tube is then placed to the bottom of the cavity and packed round with iodoform gauze. The latter should be changed every twenty-four hours, and the cavity gently irrigated, but it is wiser to leave the tube *in situ* without disturbing it, and gradually shorten its outer end as the contracting cavity pushes the tube outwards.

2. *The Submesenteric Abscess*.—An incision is made over the maximum spot of dullness which, when the patient is well under the influence of chloroform, is generally found to be near the middle line, so that in most of these abscesses a median incision is the best.

The general cavity of the peritoneum is usually opened up, but there may be some slight adhesions. Having carefully plugged the wound with iodoform gauze, by gentle manipulation an entry is made with the finger into the abscess cavity, which should be at once flushed out and then thoroughly sponged out so that as little as possible is left behind. A drainage tube is then inserted packed round with gauze, and if the condition of the patient is favourable the dressing may be left for two days without being changed so that adhesions may become definitely formed. Providing precaution is taken against the spreading of the pus into the general peritoneal cavity both during the operation and afterwards, these cases do remarkably well.

*Abscess in the Pelvis*.—These are very difficult to drain from any abdominal incision, and may be better reached through the ischio-rectal fossa on the right side. An incision is made midway between the anus and the ischial tuberosity, and the tissue of the fossa is thoroughly incised as far as the levator ani. With gentle pressure from above the bulging of the abscess may be felt, and the muscle should be perforated with a pair of sinus forceps. The opening may then be enlarged, the pus washed out, and a drainage tube inserted. I have performed this operation in one patient with an excellent result, the abscess closing in a remarkably short time. The only trouble here is the question of hæmorrhage; if a large branch of one of the hæmorrhoidal arteries or veins be divided there may be considerable hæmorrhage before it can be readily stopped.



## Tuberculous Peritonitis

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TUBERCULOUS PERITONITIS most often occurs in patients under twenty years of age, and in boys, perhaps, rather more frequently than in girls. It is an inflammation of the peritoneum associated with the presence of Koch's tubercle bacillus or the toxins produced by it.

The infection reaches the peritoneum by the alimentary canal, through the blood-stream or along the lymphatics. Tuberculous peritonitis is hardly ever primary, though it is possible for the bacilli to leave an intestine which is anatomically intact. It is generally secondary to other tuberculous foci either in the bronchial glands, the bones, joints, or even the genito-urinary tract. The mesenteric glands may be the seat of tuberculous deposits, but the more common enlargements of these glands occurring in *tabes mesenterica*, known as "consumption of the bowels," is nearly always associated with chronic gastro-intestinal catarrh, which is the result of improper feeding and is not tuberculous in origin. Anything which lowers the resisting power of the peritoneum will, as in other tuberculous lesions, determine the deposit of the bacillus, so that injuries to the abdomen, new growths in the form of cysts or hydatids, and such abdominal operations as the radical cure of hernia, etc., may lead to an attack of tuberculous peritonitis in persons who are otherwise predisposed to the disease.

VARIETIES.—Tuberculous peritonitis occurs in a miliary or *acute* form, and in a *chronic* ulcerating and fibrous type. But this classification is only adopted for descriptive purposes, since every case which is examined carefully will show evidence of the coexistence of each variety. A tuberculous peritonitis may run its course without suppuration, as a pure culture of tubercle bacillus is not pyogenic. The infection, on the other hand, is usually a mixture of tubercle bacilli with staphylococci, so that pus is often found.

*Miliary peritoneal tuberculosis* is usually acute, and partly in consequence of the intimate ancestral connection between the peritoneum and pleura, partly owing to the greater absorptive power possessed by the peritoneum covering the diaphragm, the pleura is nearly always affected in this form of peritonitis.

*Symptoms.*—The onset is generally slow, though in some cases it is so sudden that the abdomen has been opened for the relief of acute intestinal obstruction. The usual symptoms are pain and tenderness over the abdomen, a flatulent distension of the intestines, malaise, headache, quick breathing, and an anxious expression. The bowels are relaxed and there are occasional attacks of diarrhoea, whilst the patient has a hectic tempera-



ture, that is to say, the feverishness is better marked in the afternoon and evening than in the morning, and he loses flesh.

The local abdominal symptoms are often masked by those belonging to the general tuberculosis of which they form a part. But there is no doubt that many cases have been mistaken for typhoid fever until the appearance of dropsy or a post-mortem examination has revealed the true nature of the disease. The acute cases last from one to three months, and end either in death or by passing into one of the more chronic forms. When death occurs it is due more to the general condition of the patient than to the peritonitis.

The prognosis, therefore, though bad, is not quite hopeless, but it should be remembered that the condition is one which is peculiarly likely to recur. The general peritoneal cavity remains open during the acute stage of miliary tuberculosis, and is not shut off by adhesions, and the inflamed visceral layer of the peritoneum is studded with numberless small, grey translucent tubercles which feel quite hard. The mesenteric glands, though enlarged, are not greatly affected.

*Treatment.*—Unfortunately very little can be done for those cases of acute miliary tuberculosis. The affection is only part of a general disease, and it is useless to open the abdomen. Reliance must therefore be placed on constitutional remedies, whilst the open air treatment and forced feeding may be adopted as early as possible if the symptoms show any tendency to become subacute or chronic.

*Chronic tuberculous peritonitis* includes an ulcerating form in which the tuberculous deposits show a tendency to caseate, and a fibrous form in which numerous adhesions are formed.

The onset of these forms of chronic tuberculous peritonitis is usually so gradual as to pass unnoticed. The patient becomes languid, loses flesh, has a hectic temperature, and only complains of tympanites and a little abdominal tenderness. The abdomen, if there be ascites, gradually enlarges until it is rounded and shiny with many dilated veins on its surface. The cause of the ascites is not fully known. It may disappear spontaneously, but its reappearance does not seem to be always coincident with a recrudescence of peritoneal tubercle, and it is not necessarily most abundant in cases where the liver changes are most marked. The fluid is either free in the peritoneal cavity or it is localised. When it is free, the abdomen, as in ordinary ascites, is dull on percussion in front and resonant in the flanks, the line of dulness altering with the position of the patient. When the fluid is localised the cysts can be detected as tense swellings which are usually situated above the umbilicus. The caseating masses of tubercle can often be felt as nodules within the abdomen.

The disease in its fibrous form lasts months or years, and shows a tendency to undergo spontaneous cure, but in the caseating form the patient usually dies of lardaceous disease unless he is carried off by meningitis or the exhaustion produced by diarrhœa.

*Complications.*—A latent pleurisy, secondary to inflammation of the peritoneum, is the most common complication of chronic tuberculous peritonitis. A transient meningitis is sometimes observed, characterised by violent headache, vomiting, slowness of the pulse, and some retraction of the head. Sometimes the liver is profoundly affected, the cells undergoing fatty degeneration. Fæcal fistulæ, too, may be formed by the tuberculous inflammation spreading inwards through the bowel wall, or, more rarely, outwards from ulceration of the intestine. These fistulæ may discharge themselves at the umbilicus in the unopened abdomen, though they usually



appear at the scar when an abdominal section has been previously performed. They are usually of bad omen, but they sometimes close spontaneously.

I have lately had a boy under my care who suffered from most of the sequelæ of tuberculous peritonitis, and as his case is fairly typical, it may be stated briefly. He was a school-boy, aged six years, who had never enjoyed very good health, but for the week before he was admitted into St. Bartholomew's Hospital he had been more poorly than usual. He vomited at intervals and complained of severe stomach-ache. He had been obstinately constipated, but never to the extent of complete obstruction.

On his admission to the hospital on 5th July 1901 he was in obvious pain, the stomach-ache being limited to the left side of his abdomen. The abdomen was full, but was not markedly distended, and the right rectus abdominis was less rigid than the left. A rectal examination showed that the anterior wall of the rectum was somewhat bulged backwards, but no definite tumour could be felt.

During the two days following his admission to the hospital the patient's condition became worse, the abdomen was more distended, the pain increased, and there was a considerable amount of intestinal obstruction.

We made a diagnosis of tuberculous peritonitis leading to the formation of bands of adhesion. I opened the abdomen in the middle line below the umbilicus by an incision two inches in length. The coils of small intestine were adherent to each other and to the parietal layer of the peritoneum, and a thicker band of fibrous tissue appeared to be constricting a loop of small intestine just opposite the centre of the incision. I divided this band, gently separated the adhesions, and then passed my finger downwards first into the right iliac fossa to exclude the possibility of an appendicitis, afterwards into the left iliac fossa where the patient had complained of most pain. Nothing abnormal was felt on the right side, but as soon as the coils of intestine on the left side were disturbed, a gush of stinking pus escaped to the amount of four or five ounces. I allowed as much of the pus to escape as possible, and then passed a large rubber drainage-tube deeply into the iliac fossa, partly closed the wound with sutures of silkworm gut, and dressed it with cyanide gauze.

The patient did well from the 18th to the 25th July, when the symptoms of intestinal obstruction again occurred. I therefore reopened the wound and found numerous adhesions binding together several coils of small intestine. I freed these adhesions, and assured myself that there was no longer any pus pent up in the peritoneal cavity.

Three days after the operation he had a faecal fistula, for the discharge from the abdomen was distinctly faecal, and charcoal given by the mouth appeared at the wound in five hours. This fistula closed spontaneously, the faeces ceasing to pass on 22nd August. He left the hospital fat and well on 1st September.

*Treatment.*—The ordinary palliative means must first be tried in every case of chronic tuberculous peritonitis, and if these fail, an abdominal section yields the most satisfactory results if the patients submitted to operation are selected with moderate care.

The operation was first done in 1862 by Spencer Wells, who opened an abdomen for the removal of an ovarian tumour, only to find that the peritoneum was studded with miliary tubercles, and its cavity contained a large quantity of opalescent fluid. The operation has since been performed repeatedly, and the latest statistics show that 70 to 80 per cent of the cases of tuberculous peritonitis are cured by this means, even when five



years are allowed to elapse without recurrence before a patient is said to be cured.

The mortality from the operation is not more than 3 per cent, and the cases which are not cured are markedly relieved.

Recurrence takes place from time to time, and in the ascitic form the abdomen may refill. Abdominal section may then be repeated and with successful results, but in a few cases I have seen a tuberculous dermatitis spreading from the edge of the wound.

The pathology of the cure is not understood, though it is supposed that the abdominal section excites so lasting an hyperæmia of the peritoneum as to check the tubercular invasion.

The best results are obtained in the ascitic forms where there are but few adhesions; the least successful cases are those of miliary tubercle.

Abdominal section is useless in patients who are suffering from general tuberculosis, extensive lesions of the lungs, tuberculosis of the liver, kidney, intestines, and female generative organs, though in the slighter cases the infected organ may be removed at the time the fluid is evacuated.

The incision in the ordinary ascitic cases should be made below the umbilicus, care being taken not to wound the bowel or omentum, for they are often adherent to the peritoneum; not to injure the bladder which in children often extends higher in the abdomen than is expected, and not to work too cautiously by dissecting with undue care through the thickened and cedematous subperitoneal tissues. These tissues are sometimes so greatly thickened in chronic tuberculous peritonitis as to be mistaken for the omentum, and an extensive dissection, by opening up different layers of tissue, militates against rapid healing of the wound. The peritoneum should be cautiously pricked with a scalpel after it has been raised by forceps, and the incision should be enlarged with blunt-pointed scissors to the full extent of the wound, the fingers being used as a director to prevent injury to the bowel. The fluid should be allowed to escape freely, but the adhesions must be treated according to the individual case. The only general rule that can be laid down is to touch them as gently as possible, for they may bleed freely, and the hæmorrhage is difficult to stop, and to do no more to them than is absolutely necessary. Even dense adhesions have been known to disappear completely after a simple abdominal section.

I prefer not to flush the abdominal cavity, especially if there has been a localised abscess, as the cases do equally well without it, and there is some danger of disseminating the disease still more widely. Hot salt solution or boric lotion should be used when it is decided to flush the peritoneum, and no antiseptic, however weak, for it is impossible to remove it all from the abdominal cavity, and many pints are used if the infusion has been large. This was impressed upon me a year or two ago, when a patient from whom I had removed twenty quarts of tuberculous ascitic fluid had a sharp attack of mercurial poisoning, because I had flushed his abdomen with a 1 in 10,000 solution of biniodide of mercury.

The abdominal wound should be sewn up at once if the effusion has been clear or only sero-purulent, but it must be drained freely if an abscess has been opened. All parts of the wound must be brought into good apposition, for there is some tendency to the formation of a ventral hernia after these operations. The drainage-tube should be removed at the end of two or three days.

The after treatment, upon which much of the success of the operation depends, consists in keeping the patient quiet, but not necessarily motion-



less, in bed, and in a room whose temperature is maintained at 65° F. Small doses of opium may be given for the first day or two after the operation if there is much pain; if the temperature rises and enemata fail to act, a little magnesia or a few grains of calomel will reduce it by relieving the bowels. The food should be given in a concentrated form, and it may often be peptonised with advantage. A belt should be worn for a considerable time after the end of convalescence, in order still further to reduce the risk of a ventral hernia.

PERITONITIS ASSOCIATED WITH PNEUMOCOCCI.—Tuberculous peritonitis is likely to be mistaken for another insidious form of peritonitis associated with the presence of pneumococci.

The affection exists in two forms, either primarily, when it may coexist with meningitis, pleurisy, or pericarditis, or secondarily in connection with pneumonia.

The disease runs a prolonged course, characterised by great pain, much meteorism, and constant vomiting. The bowels may remain regular throughout the attack, and there is often a normal temperature.

The peritonitis is always suppurative, the pus being of the consistency of cream, greenish in colour, and with a tendency to clot. It has a faint and characteristic smell. Laparotomy affords the only prospect of successful treatment, and the sooner it is done the better, for the plastic exudation glues the intestines together and makes the operation difficult if it be delayed.

The surgeon must be especially careful not to overlook abscesses in other parts of the serous cavities, as pneumococcic peritonitis is often associated with empyema and suppurative pericarditis.

SUPPURATIVE PERITONITIS ASSOCIATED WITH SUPPURATION IN OTHER SEROUS MEMBRANES, or POLYORROMENITIS.—The Italian physicians have given the name polyorromenitis to an analogous condition of suppuration in several serous cavities, and the disease has been carefully studied by Dr. Frederick Taylor.

There is but little doubt, however, that the affection is not a definite pathological entity, but is often the result of a general infection often beginning without any assignable cause, and running an abnormal course in weakly persons.

Bacteriological examination shows that tubercle bacilli, pneumococci, staphylococci, and even streptococci, are found in the pus contained in the serous cavities.

The affection is more frequent in males than in females, and the majority of patients are between the ages of sixteen and thirty, though one of my cases occurred in a girl of five and a half, and the other in a girl aged four years. One serous cavity is usually invaded before the others, and the peritoneum is generally attacked first, then the pleuræ, and the right pleura before the left. Sometimes the pleura is attacked before the peritoneum, pericarditis following the pleurisy especially, when it has been on the left side. The interval between the invasion of the different serous sacs may be a few weeks or some months.

The prognosis is fairly good. Picchini finds that in fifty cases there were twenty-two deaths, twenty-one recoveries, and seven cases improved, in the two cases under my own care the one recovered, the other died. Recovery may take place either completely or with the formation of adhesions, the fatal termination may either be directly from suppuration or more remotely from phthisis.

The treatment consists in letting out the pus as it is formed in the



different cavities, and by the use of mercurial inunctions over the affected parts, combined with the ordinary medical treatment of suppurative peritonitis (see p. 97).

PERITONITIS IN THE NEW-BORN.—*Fœtal or Intra-uterine Peritonitis*.—Peritonitis in all its forms is known to occur during foetal life, for children are born dead with evidence of ascitic, adhesive and suppurative inflammation.

Syphilis is an assigned cause, probably with truth in many cases, but that there are other reasons for the peritonitis is shown by the fact that of twins one may be born alive and healthy, the other dead with advanced peritonitis.

The chronic peritonitis of foetal life is often associated with, and perhaps causes, an arrest in the development of the alimentary canal. I had such a case under my care on 7th December 1900, when a male child aged four days was brought to the Victoria Hospital for children because the bowels had not been relieved since his birth. On passing a finger into the rectum a distinct membrane could be felt stretching across the bowel about  $2\frac{1}{2}$  inches from the anus. The obstruction was considered to be due to an imperforate rectum, and the child was submitted to a left inguinal colotomy, but without much relief of symptoms, and he died seventeen hours after the operation. The autopsy showed that the bulk of the intestines were matted together to form a roundish ball. The adhesions were of long standing, and must have resulted from intra-uterine peritonitis. The intestine ended blindly about two feet below the stomach, and at a second point situated on the right side of the abdominal cavity. The bowel was almost black in colour: it was covered with lymph and presented many adhesions.

*Acquired Peritonitis*.—Peritonitis is also found as an acquired disease in the new-born. It usually depends upon puerperal infection, and is brought about by the direct extension of septic inflammatory processes from the umbilical cord. It is due in a few cases to rupture of the bowel resulting from an imperforate anus, or still more rarely to rupture of the sigmoid flexure which has been assumed by Zillner and Gersich to be due to direct pressure upon the bowel during parturition. I have also seen it start from the sloughing of the thin wall of a true exomphalos.

The symptoms are often obscure, and the condition may not be recognised during life. But in a typical case the onset is sudden, with vomiting and a temperature of  $103^{\circ}$  to  $105^{\circ}$  F. The pulse is small and rapid, the respirations hurried, and there is great prostration. The abdomen becomes swollen and tympanitic, and there may or may not be diarrhoea.

The prognosis is very bad as nearly all the cases die.

*Treatment*.—Abdominal section is useless, but the stomach should be fomented with compresses soaked in hot boric lotion. Drop doses of paregoric may be given every hour with ten drops of brandy in a teaspoonful of hot water. The child should be suckled, but if this is impossible it should be fed on small doses of peptonised food or raw meat juice.

Post-mortem examination shows that the inflammation has been either local or general. The local inflammation is generally limited to the neighbourhood of the umbilicus or the liver. The infection is usually streptococcic.

PERITONITIS DUE TO ACTINOMYCOSIS.—Localised peritonitis is occasionally met with as a result of the invasion of actinomyces—the Ray fungus.

The primary seat of growth is the alimentary tract or the liver, the appendix and cæcum being the particular parts of the intestine most often attacked.



When the growth occurs at the right iliac region there appears to be no means of distinguishing it from an ordinary suppurative appendicitis. It runs a much more chronic course, however, for when the abscess has been opened a fistulous tract is left, the skin becomes involved in the inflammatory process, and the diagnosis is made at last by finding that the pus contains the characteristic actinomycotic granules.

When the liver is attacked the symptoms are still more obscure, for it may be impossible to detect any abscess, though there is evidence of extensive inflammatory thickening of the affected organ and of the overlying peritoneum. There is usually very extensive suppuration, and the patient dies with abscesses in many of his organs, some of the pus being the result of dissemination of the actinomycetes.

Large doses of iodide of potassium have a very remarkable effect in curing actinomycotic inflammations, and even when they do not cure the condition of the patient is greatly improved. The drug may be rapidly increased from the ordinary medicinal dose of five or ten grains until a drachm is given three times a day. The dose is then reduced to forty or fifty grains three times a day, which may be given for many weeks in succession. The patient actually gains in flesh and spirits so long as he is taking these large doses of potassium iodide until he ceases to need the drug.

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PERITONEAL tumours can be classified, like tumours in other parts of the body, into innocent and malignant, each group containing solid and cystic swellings.

*The solid innocent tumours* of the peritoneum are either inflammatory, or they grow in virtue of the peritoneum being a mesoblastic structure, and they are therefore of the mesoblastic type.

*The inflammatory swellings* are always subperitoneal, and occur in the loose cellular tissue which forms the lower or subphrenic portion of the vast and complicated distribution of mesoblastic elements primarily developed round the great vessels in front of the spine, and afterwards enclosing the receptaculum chyli and its glands and branches, the great sympathetic plexuses, and the lumbar and sacral nerves. It is best known at the part where it is prolonged into the liver, as Glisson's capsule, and where a more condensed portion with an additional quantity of fat forms the capsule of the kidney. The less known portions cover the pancreas, the third part of the duodenum, and the presacral region.



The subperitoneal tissue is subject to many causes of inflammation, some of which have already been described under the headings of "Subphrenic Abscess," "Perinephric Abscess," "Pelvic and Prevesical Cellulitis," and "Fat Necrosis" associated with disease of the pancreas. Many idiopathic forms exist which resemble the "cold abscesses" of the old pathologists. These abscesses may appear spontaneously in delicate persons with a predisposition to tubercle. They are found after slight injuries, and at the present time I have a man under my care with a lumbar abscess which is traceable to a carbuncle on the neck from which he suffered some two months since. Similar metastatic abscesses may occur after such operations as the radical cure of hernia, castration, or ligature of a varicocele when the surgeon from any cause has been unsuccessful in keeping the wound aseptic.

*The treatment* in many of the slighter cases consists in fomenting the part and keeping the patient at rest for a few days, when they subside spontaneously; but when suppuration has occurred the abscess should be laid open, scraped with a flushing scoop through which boracic lotion is flowing at a temperature of 110°-115° F., and afterwards sewn up to obtain union by first intention.

The chief innocent peritoneal tumours are fibromata, lipomata, and a mixture of the two, or fibro-lipomata. They usually grow from the subperitoneal tissue, but they are occasionally found within the peritoneal cavity springing from the mesentery.

*The fibromata* are of pathological rather than of surgical interest. They are usually situated on the visceral aspect of the parietal layer of the peritoneum, in the region of the liver and spleen, either as hard plates or small globular masses, and they are associated with long-standing inflammation of the hepatic and splenic capsules. A superficial examination shows that they resemble cartilage in their physical characters, but histology proves them to be masses of dense fibrous tissue, like that of the *substantia propria* of the cornea, provided with flattened connective tissue corpuscles. They often become calcified. There is no treatment for them, as they are hardly ever recognised during life.

*The fatty tumours* always grow from the subperitoneal tissue. To a certain extent they are normal structures, and every surgeon who performs the radical operation for the cure of hernia knows that he often meets with them at the internal abdominal ring in males and in the femoral canal in women. They are also found at the obturator foramen and in the linea alba, and in all these situations they may be mistaken for herniæ. Lipomata sometimes grow in the subserous tissue behind the kidneys and in the iliac fossa, whilst by insinuating themselves between the layers of the mesentery they may encircle the small intestine, and may thus appear to be intraperitoneal in origin. Indeed, in some cases they have been found to lie free in the abdominal cavity, the explanation of such a condition being that they have been attached by a very delicate pedicle, which has been torn through either shortly before death or during the post-mortem examination.

*The lipomata* or fibro-lipomata are usually of small size, but sometimes they attain huge dimensions, and have been known to weigh sixty pounds. Histologically they are pure fatty tumours, fatty tumours with much fibrous tissue or myxomatous growths.

The symptoms vary with the situation and size of the growth. Lipomata lying in the inguinal and femoral rings, even when they are small, have been mistaken for inflamed or strangulated herniæ. The resemblance is often close, because the tumours give an impulse on coughing, and may



cause both pain and vomiting. The larger growths have often been diagnosed as ascites and ovarian tumours. These mistakes, however, may be avoided with a little care, for the smaller tumours are situated more superficially than a true hernia, and the larger lipomata are soft, and move, as a whole, in a very characteristic manner.

The prognosis in the small tumours is favourable, and they may be removed without hesitation if they are troublesome. But in the large tumours the prognosis is much less favourable. They are deeply seated in the abdominal cavity, a large operation is required for their removal, and they have often formed dense adhesions to the surrounding tissues and organs. MM. Terrier and Guillemin have only had four recoveries in eleven operations.

*The myomata* spring chiefly from the uterus and its ligaments, but they occasionally grow independently from the scattered muscular fibres in the broad ligament, or from the suspensory bands in the mesentery and elsewhere.

The fibro-myomata occur as a variety of these unstripped muscle tumours, and they are sometimes multiple, as in the case quoted by the late Mr. William Anderson, which occurred at Tokyo Charity Hospital. The patient was a girl aged nine years, from whom twenty-one rounded tumours of various sizes were removed from different parts of the subperitoneal tissue, extending from the pelvis to the hypochondrium. The heaviest tumour weighed more than seven pounds. They were soft, but elastic, white and waxlike on section, and under the microscope they were found to consist of dense fibrous tissue containing some unstripped muscle fibres. The tumours were distinctly encapsuled, and shelled out readily. But the patient, who was an emaciated child, died of shock directly after the operation.

*The innocent cystic tumours* of the peritoneum are the result of injuries; they are inflammatory, parasitic, or connected with foetal remains.

*Lymphangiomata and hæmatomata* are found within the peritoneal cavity in consequence of obstruction to the lymphatics or the blood-vessels, especially in the immediate neighbourhood of new growths. They are not of any practical importance. Dr. Rolleston has called attention in the *Transactions of the Pathological Society of London*, vol. xlix. 1898, p. 145, to the manner in which cysts may be produced by simple obstruction in the lesser sac of the peritoneum. The case was that of a man aged twenty-five, who was seized with vomiting a fortnight before his death, the vomiting continuing during the rest of his life. He had pain in the lower part of his abdomen which was never very acute. It came on before he vomited, and was sometimes relieved by the emptying of his stomach. The patient had always suffered from constipation, which had been more marked since the onset of his illness. There was no history of any abdominal injury, there was no jaundice, hæmatemesis, nor melæna, and the patient had wasted considerably since the vomiting began. An indistinct tumour was felt below the left hypochondrium, with the stomach resonance above it, the heart's apex being in the fourth costal interspace. The diagnosis lay between carcinoma of the stomach and a pancreatic cyst. It was found at the autopsy that the patient had died of an acute inflammation of the pancreas. There was no general peritonitis, but the lesser sac of the peritoneum (the "omental bursa" of American authors) was found distended with grumous fluid, yellowish brown in colour, and turbid. The foramen of Winslow was obliterated, so that there was a localised peritoneal effusion, the stomach being pressed forwards and flattened on the front of the distended lesser sac, the walls of which were thickened by inflammation and showed scattered



areas of fat necrosis. The mechanism by which the effusion becomes limited to the lesser sac depends on the sealing up of the foramen of Winslow, which may be one of the first effects of a pancreatic inflammation, or may be caused by a pre-existing inflammation spreading from the common bile-duct. Carcinoma, in like manner, may obliterate the foramen of Winslow by adhesions starting from the gall bladder.

Multiple cysts filled with serous fluid are sometimes developed in the root of the mesentery without invading the pelvis.

The parasitic cysts are *hydatids*. They may be sterile cysts—"acephalo-cyst"—the ordinary proliferating cyst, or proliferating cysts in which the secondary cysts are undergoing degenerative changes.

The following case shows the ordinary symptoms and treatment of a case of peritoneal hydatid. A boy of 16 was admitted to St. Bartholomew's Hospital for a tumour which occupied the whole of the lower half of the abdomen from the umbilicus to the symphysis pubis in its long diameter, and from crest to crest of the ilium transversely. The tumour was divided sharply into two lobes by a constriction which was apparently due to the urachus. The swelling was fixed, and the skin was movable freely over it. There was no thrill, but it was dull on percussion. Abdominal exploration showed the tumour to be a sterile hydatid cyst which grew from the left side of the pelvis, and was adherent to the omentum, intestines, and bladder. As it was impossible to separate the external or adventitious cyst from these adhesions the tumour was opened, the fluid was allowed to escape, and the whole of the endocyst was removed. The edges of the adventitious cyst were then stitched to the abdominal walls, excluding the skin, a drainage tube was inserted to the bottom of the cyst, and the skin was closed over the wound. The tube was allowed to remain from the 11th to the 21st September when it was removed, and the patient was discharged with the wound soundly healed on October 8th. The ectocyst may often be closed at once if the surgeon is sure of his asepsis, and the abdominal wound will then heal by first intention.

*Cysts due to foetal remains* are usually found in the tissue between the rectum and coccyx. In the simplest form they are single or multiple cysts lined with cylindrical ciliated epithelium and containing a clear serous fluid. An instance of such a tumour was recently under observation at the Victoria Hospital for Children, in a child aged two months, who was suffering from retention of urine and obstruction of the bowels. A bimanual examination through the rectum and abdomen revealed a tense and elastic swelling upon the right side of the pelvis reaching from the pubes to the umbilicus. The swelling was fixed and pyriform in shape, the upper broad end being rounded and sharply defined. The abdomen was opened in the middle line, but the tumour was too deeply seated and firmly fixed to allow of its removal. The child died two days after the operation, and the kidneys were then seen to be in a condition of hydronephrosis, the ureters were dilated, and the bladder thickened from the pressure exercised by the swelling. The rectum was normal for an inch above the anus, but immediately beyond this point a large oval swelling projected into the lumen without causing any gross lesion. The uterus, ovaries, and Fallopian tubes were normal. The tumour was a simple, thick-walled sac, filled with clear fluid, and there is little doubt that the child would have lived if I had incised the swelling and let out the contents. The specimen is in the museum of St. Bartholomew's Hospital.

*Dermoids* are sometimes intraperitoneal, though they are more often found in the presacral region. The intraperitoneal dermoids may occur as



multiple tumours of small size attached to the peritoneum, hanging from the under-surface of the liver, or embedded in adhesions between the coils of intestine.

The presacral dermoids are usually found in women, probably because their pelvic organs are more often and more carefully examined than those of men. A few months ago I saw one in an art student, aged 21, in whom it would probably have remained undetected if he had not suffered from an attack of appendicitis which led to a rectal examination. He was then found to have an elastic tumour as large as a hen's egg in the tissue behind the centre of the posterior wall of the rectum, just above the internal sphincter. The swelling was tender, and for a time it was thought to be inflammatory in origin and connected with the appendicular trouble from which he was suffering. The patient recovered in due course from the attack of appendicitis, but the tumour remained unchanged, and an exploration was carried out on the assumption that it was a chronic abscess which might end in a fistula. The swelling was incised through the ischio-rectal fossa, and its contents immediately showed it to be a dermoid cyst. The wound was therefore cleansed and closed, as it was placed inconveniently for the larger operation which had now become necessary. The patient was then turned on his side, the coccyx was removed, and the entire cyst was dissected out. The man recovered without delay, as the wound healed by first intention.

*Nævi* of the peritoneum are rare, but a case is recorded by Mr. Arbuthnot Lane in a child aged seven years. The tumour had been noticed at birth, and had increased in size until it formed a prominent swelling occupying the right side of the abdomen. It extended from near the middle line in front to the loin behind, and from the margin of the thorax above to the iliac crest. The abdominal wall seemed to be involved, and fluctuation was felt over the most prominent portions. In the course of a successful excision of the growth several large cysts, some nearly as big as an orange and many smaller ones, were opened and found to contain liquid blood, their inner aspect being smooth and polished. The tumour appeared to start in the outer surface of the peritoneum, forming livid projections into the peritoneal cavity, and externally it invaded the muscles of the abdominal wall. It extended upwards beneath the diaphragm and backwards into relation with the kidney. It proved to be *nævroid* in character, and was apparently undergoing rapid degeneration.

MALIGNANT DISEASE OF THE PERITONEUM still stands in need of much further study from a pathological standpoint. It is divisible broadly into sarcoma and carcinoma, but the two groups are not yet clearly differentiated from each other, and their tumours are sometimes mistaken for inflammatory tumours which either disappear spontaneously or when a surgeon has been led to give a fatal prognosis after an exploratory abdominal section.

*The sarcomata* are either primary, arising in the retro-peritoneal tissue, the great omentum, the mesentery, or the broad ligaments; or they are secondary to disease of the testis or ovary, from which they spread by direct extension. They occur more often in males than in females, and in adults than in children. The peritoneal sarcomata are usually described as being of the round or spindle-celled variety, or as lympho-sarcomata, but there is an increasing tendency amongst pathologists to classify most of these tumours as endotheliomata.

*The endotheliomata* are generally seen as multiple flattened growths, whitish in colour, and either discrete or connected with each other by bands of adhesion. The growths usually cause effusion into



the peritoneal cavity, and they give rise to secondary deposits in other serous membranes, though the various organs of the body usually escape. Endothelial tumours are characterised by the presence of nests or clusters of cells which have a columnar appearance at the periphery of the growths. These clusters of cells lie in a dense fibrous tissue and exactly follow the course of the lymphatic vessels. The origin of the cells is not yet known accurately, but they seem to be derived from the endothelial lining of the lymphatics unless they come from the multiplication of the epithelium of the coelom or primitive body cavity.

Peritoneal endotheliomata, like endotheliomata in other parts, undergo various degenerative changes, chief amongst which are their conversion into myomata and the formation of blood cysts by an angioplastic formation.

*Carcinomata* of the peritoneum are all secondary, the primary growth being found in the gall bladder, pancreas, or ovaries. Enormous masses of "colloid cancer" are sometimes found invading the mesentery, probably by extension from the stomach or rectum.

The symptoms of malignant disease of the peritoneum are always obscure. When it is primary there is probably very little more than loss of weight, "dyspepsia," and general malaise. The discovery of an abdominal tumour, which increases in size and causes various pressure symptoms according to its position, sometimes renders the diagnosis more clear, though it occasionally makes it more obscure; for in several cases an abdominal section has revealed the presence of a swelling which was thought to be a sarcoma, so large and so intimately adherent to the surrounding tissues and organs as to be beyond the possibility of removal. Yet from the time of the operation the swelling has got steadily smaller, and in the end has completely disappeared, the patient recovering his former good health. I have ventured to speak of these swellings as "Vanishing Tumours" (*The Lancet*, vol. i. 1899, p. 583). They were probably retro-peritoneal inflammatory swellings, connected perhaps with tubercle, syphilis, or sepsis, which have been diagnosed incorrectly as malignant.

No treatment is of much avail in cases of malignant disease of the peritoneum. Surgery offers no radical cure, and medicine only affords the palliative measures which ensure a euthanasia. Endothelial tumours can usually be shelled out of the capsule in which they are enclosed. A determined attempt should therefore be made to enucleate them in spite of the adhesions of surrounding organs and tissues to their exterior, for if they are left untouched they soon cause death.

LITERATURE.—WILLIAM ANDERSON. "A Discussion on the Surgery of the Sub-peritoneal Tissue," *The British Medical Journal*, vol. ii. 1896, p. 1087.—D'ARCY POWER. "Some interesting Abdominal Cases," *The St. Bartholomew's Hospital Reports*, vol. xxxvii. 1901.

**Perityphlitis.** See APPENDIX.

**Pertussis.** See WHOOPING COUGH.

**Petechiæ** are small crimson or purple spots met with in the cutaneous or mucous surfaces of the body, due to the transudation of the colouring matters of the blood from the capillaries into the adjacent tissues. They must be differentiated from simple hyperæmia, and also from flea-bites. Hyperæmia is distinguished by the colour fading on pressure, whereas in petechia it remains, though perhaps in a less degree. A flea-bite may be distinguished by observing the central puncture due to the



bite. In themselves petechiæ have no clinical importance, and call for little comment and less treatment. But they have significance from the stand-point of the toxæmia, which is invariably their cause. Their presence indicates weakened or ruptured capillaries, and this weakening or rupture is significant of the action of toxic substances in the capillary wall, and the questions that present themselves to the practitioner are—(1) What relationship, if any, do these small visible hæmorrhages bear to the various symptoms from which the patient suffers? (2) May the symptoms of a nervous or other nature be explained by the evidence of a similar weakening of the vessels in the central nervous system or elsewhere, and resulting failure of nutrition in the special tissue involved. Looked at from this stand-point petechiæ may furnish assistance in diagnosis, may guide to prognosis, and be helpful in treatment.

The occurrence of petechiæ and very acute toxæmiæ, *e.g.* typhus, will be referred to under their special headings.

**Petit Mal.** See EPILEPSY.

**Phagocytosis.** See LEUCOCYTOSIS, IMMUNITY.

**Phantom Tumour.** See TUMOURS.

## Pharynx.

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### Examination of the Pharynx, etc.

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The patient should be seated in a chair fitted with a head rest. A light is placed on his left side slightly behind him, and on a level with his eyes. During the examination the surgeon may stand, or be seated at the patient's right side, slightly in front of him, and facing in the opposite direction. By means of a forehead mirror, which is worn over the right eye, light is reflected into the patient's mouth.

The patient is directed to keep the tongue as flat as possible, while its tip lies behind the lower incisors, and to breathe quietly and regularly through the nose. The sides and middle of the tongue are then successively depressed by means of a suitable instrument—preferably Fränkel's tongue-depressor.

This simple procedure excites in some persons retching, so that a satisfactory view is unobtainable. The following precautions should be taken in dealing with such patients:—The mouth should not be opened too widely; the breathing should be shallow and regular; and the point of the tongue-depressor should not be placed too far back on the tongue. If these measures fail, the patient himself should try to use the tongue-depressor effectively. Türk's is the most suitable for this purpose. The examination may be more successful at a subsequent visit after the patient's nervousness has passed off.



The normal pharyngeal mucous membrane is pink and moist ; there may, however, be considerable variations in both respects without any accompanying discomfort. On the other hand, a patient who is not neurotic may complain of "sore throat," although no abnormal appearance is discoverable. The colour is best observed on the soft palate and posterior wall. It should be noted that when the mouth is first opened the pharynx occasionally presents a marked vascular injection which passes off in a few seconds. Variations in the amount and character of the secretion are most manifest on the posterior wall. Excessive mucus or muco-pus, coming partly from the naso-pharynx, may be here seen sliding downwards. Undue dryness may, on the contrary, be the prominent feature, the secretion forming a thin varnish-like coating or a thick tough discoloured crust.

Besides the colour of the soft palate, its mobility should be observed while the patient says "Ah!" also its distance from the posterior wall. A glance should be taken at the length of the uvula. The size of the tonsils, their relations to the anterior and posterior faucial pillars, and the number and contents of their crypts should be studied. The condition of the lateral walls, especially as regards the presence of granulations, is to be noted. Lastly, the posterior wall claims attention with reference to its colour, secretion, the presence of small vascular twigs which show through the mucous membrane, or occasionally of a large pulsating prominence which is caused by a bend of the internal carotid. In examining this region it is important that the neck be not twisted, otherwise one half of the posterior wall (on the side towards which the patient faces) will be shallower than the other half, and by the uninitiated may be mistaken for an abnormal bulging, *e.g.* retro-pharyngeal abscess. The examination of the pharynx may be completed by pushing the tongue-depressor far back and causing the patient to retch ; in this way more of the posterior wall both above and below is brought into view, possibly also the epiglottis, while the lateral walls become prominent, and the tonsils partly escape from between the faucial pillars. Probing will be facilitated by painting or spraying the region to be examined with a cocaine solution (10 per cent).

Children who cannot be coaxed to open the mouth may be forced to adopt buccal respiration by pinching the nose. If they still persist in clenching the teeth, the tongue-depressor should be introduced behind the last molars and the pharynx touched. Retching will be thus induced, and the mouth opened of necessity.

**ANÆMIA.** — In general anæmia the pharyngeal mucous membrane, especially that of the soft palate, appears unduly pale. The pallor in this region commonly observed in tuberculosis is probably only present after general anæmia has become established, and is therefore of no special diagnostic value.

**HÆMORRHAGES.** — Pharyngeal hæmorrhage may take place in the course of certain diseases in which the constitution of the blood or the vessel walls have undergone change, *e.g.* hæmophilia, purpura, leukæmia, scurvy ; or which, by causing a general stasis, favour its occurrence, *e.g.* emphysema, cardiac insufficiency, cirrhosis of the liver, granular kidney. On the other hand, the bleeding may be of purely local origin, and due to operation, suppuration, or ulceration. Those with varicose veins at the base of the tongue, or on the posterior wall of the pharynx, are said to be liable to occasional slight hæmorrhages, especially if they suffer from pharyngitis sicca, necessitating frequent hawking.

Bleeding may also take place into the mucous membrane and produce ecchymosis, or even a hæmatoma, of which a few cases have been reported.



The amount of blood lost may vary from a few drops to what proves fatal, as in the erosion of a large artery.

The source of a moderate hæmorrhage can usually be found at once. This is not so, however, when the bleeding is due to no known cause, and is scanty or has ceased. In cases of this kind the hæmorrhage, especially when recurrent, readily arouses suspicion of lung disease, and it is then necessary to satisfy the patient, or possibly an insurance company, as to the place of origin. The possibility of the bleeding being from the nose, naso-pharynx, or gums, should be remembered. The examination of the lungs, and the consideration of the patient's present and past state and family history, usually settle the question of hæmoptysis. Occasionally, however, they prove negative, or merely afford grounds for surmise. It is then necessary to compare the features of a pharyngeal with those of a pulmonary hæmorrhage. In doing so, weight must not be attached to the patient's sensations, which often lead him to the erroneous belief that the blood comes from the throat. In a pharyngeal hæmorrhage the amount of blood expectorated must be scanty, otherwise the bleeding point will be discovered; further, it is hawked, not coughed up. When from the lungs usually the sputum is liquid, florid, and frothy, the blood is intimately mixed with the mucus, and tubercular bacilli are present. If the blood is expectorated in thick or partially dry clots, it comes from the upper air tract, or has been present there for some time. The blood-stained spit that follows the detachment of crusts in pharyngitis sicca contains pavement epithelium. If blood is seen in the trachea its source is almost certainly there or in the chest. Finally, it should be remembered that hæmoptysis is of common occurrence, while pharyngeal hæmorrhage without an apparent cause is very rare; both may, of course, co-exist. Indeed, a diagnosis of pharyngeal hæmorrhage is scarcely justifiable unless the bleeding point has been seen, or hæmorrhage produced by touching the suspected spot.

Slight bleedings may be checked by allowing pieces of ice to melt in the mouth, or by applying to the bleeding area hydrogen peroxide, liq. ferr. perchlor. or a mixture of tannic (3 parts) and gallic (1 part) acids, made into a hard paste with a little water (Hovell). For bleeding gums, a gargle of chlorate of potash or of iron alum, or the direct application of lunar caustic will prove effectual. If the source is a varicose vessel the affected portion is best destroyed by the galvano-cautery point.

When the hæmorrhage is profuse, tampons saturated with a styptic should be firmly pressed over the bleeding area. Ligature of the carotid artery has even been found necessary.

Hæmatomata call for treatment only when their size causes inconvenience; they should then be evacuated.

FOREIGN BODIES.—A great variety of foreign bodies has been found in the pharynx. The most frequently occurring are those that have been introduced with the food, *e.g.* fish bones, spicules of rabbit bones; leeches and worms also have been sucked in while drinking from pools. The food itself may constitute a foreign body, as when a large piece of meat "sticks in the throat." Objects held in the mouth may fall, or be inadvertently drawn backwards, *e.g.* false teeth, pins, needles, pieces of pipe stem. Lastly, articles of all sorts have become lodged in the throats of children and lunatics by accident or design.

Diminished sensibility of the pharyngeal mucous membrane and paresis of the constrictors favour the lodgment of extraneous substances.

When the foreign body is large it usually becomes impacted about the



upper end of the œsophagus, in one of the pyriform sinuses, or between the epiglottis and tongue. If sharp and fine it may enter the mucous membrane at any point; the tonsils, faucial and lingual, are, however, the favourite resting places, and it is from these that fish-bones are oftenest found to project.

A foreign body may be present without the patient's knowledge, but it commonly produces irritation or obstruction. When sharp, a pricking sensation is usually experienced, which may be followed by acute inflammation, swelling, and abscess. On the other hand, the object may be of such a size as to cause inability to swallow, dyspnœa, and even suffocation.

The diagnosis, as a rule, is easily made on examining the parts, if need be, with the laryngoscope; when inspection fails, palpation may succeed. Occasionally the X-rays will assist.

If no indication of a foreign body can be found, the symptoms may be due to injury caused by one which is no longer present, or may be neurotic in origin (see paræsthesia of the pharynx).

In the majority of cases the symptoms quickly pass off on removal of the extraneous substance. Hysterical and neurasthenic patients may, however, complain of a soreness for months afterwards. The possibility of the presence of a second foreign body must not be overlooked.

A dangerous issue is uncommon, but deaths have occurred from suffocation, and from perforation of the large arteries.

A foreign body long retained in a tonsillar crypt may become encrusted with mineral salts and form a calculus (tonsillith).

The treatment consists in removing the foreign body as soon as possible. This can usually be effected by means of suitable forceps. If suffocation is imminent, it may be necessary to perform tracheotomy in the first place.

PHARYNGOMYCOSIS.—*Keratosis pharyngis*.—The disease commonly but incorrectly termed "pharyngo-mycosis" is characterised by the presence of small, isolated, tough, firmly adherent, white excrescences on the healthy mucous membrane. The excrescences may appear as points, tiny patches, or elongated outgrowths, attaining a length of from 2 to 10 mm. They can usually be torn off only with difficulty, and one of the most striking features of the disease is the rapidity and persistence with which they recur after removal.

The excrescences are most commonly found on the faucial tonsils and at the base of the tongue; much less frequently on the lateral parts of the pharyngeal wall and in the naso-pharynx. In a few cases they have been observed on the epiglottis, in the larynx (Gray), and nose (Wright).

The subjects of this disease are usually between fifteen and forty years of age; females are affected oftener than males. It is doubtful whether digestive or other constitutional disturbances predispose.

A few small excrescences may occasion no symptoms. When prominent, however, they may give rise to a feeling of discomfort or irritation, which in turn may cause hawking and coughing. The patient's temperament also plays a part, so that the symptoms do not always bear a direct relation to the morbid appearances. The general health, as a rule, is unaffected.

If one of the excrescences be examined, the surface is found to be soft, while the central part is tough and firm. Microscopically, the soft substance is seen to consist of a granular material, scattered through which are numerous leptothrix filaments, while the central part is made up of closely-packed cornified epithelial cells.

The abundance of the leptothrix led the earlier investigators to believe that the disease was due to the presence of this saprophyte. The affection



consequently was believed to be a mycosis, hence the names—mycosis tonsillaris benigna (B. Fränkel), pharyngomycosis leptothricia (Heryng). Recently, however, Siebenmann has shown that the excrescences are due to an excessive cornification of the lacunar epithelium, and as a slighter form of this process goes on in the tonsillar crypts very frequently, he terms the condition "hyperkeratosis lacunaris." Brown Kelly subsequently adduced further proofs in support of Siebenmann's statements, and pointed out that, while the disease under consideration is a keratosis, a true mycosis leptothricia is occasionally observed in the pharynx.

When keratosis pharyngis is well developed the diagnosis is easy. If only a few small excrescences are present on the tonsils, they may be taken for the deposits of chronic lacunar tonsillitis, which is commonly associated with keratosis. These deposits, however, are distinguished by being soft, readily pressed out, and foul smelling.

The disease, after persisting for a varying period, sometimes for years, may pass off spontaneously.

If little or no inconvenience arises from the excrescences, it usually suffices to explain their harmless nature to the patient. On the other hand, if they are causing discomfort or mental disquietude their removal should be undertaken. Many medicinal agents—*e.g.* salicylic acid, zinc chloride, and iodine preparations—have been employed for this purpose, but as a rule nothing short of the tedious process of picking off each excrescence and cauterising its site has succeeded in eradicating the disease.

NEUROSES OF SENSATION.—*Anæsthesia and Hypæsthesia*.—Complete or partial loss of sensation, due to involvement of the glosso-pharyngeal and vagus, may be of central or peripheral origin. The chief central lesions are hæmorrhages, tumours, gummata, tabes, bulbar paralysis, syringomyelia, multiple sclerosis, and pseudo-bulbar paralysis. The conditions that may affect the nerves peripherally are diphtheria, various local inflammations, influenza, typhus, cholera, and pneumonia. Anæsthesia is not infrequently associated with hysteria, and is present in some cases of epilepsy for a short time after the seizure. In anæmia and in old age diminished sensibility is occasionally detected. Transient anæsthesia or hypæsthesia may also be artificially produced by cocaine, eucaine, menthol, ethyl chloride, ice, morphia, bromides, chloral, etc.

Anæsthesia may be partial (hypæsthesia) or complete, unilateral or bilateral, confined to the soft palate, or affecting the entire pharynx. If partial and limited, it may escape the patient's notice. If marked and extensive, deglutition will be incommoded by the inability to feel the bolus of food in the throat, and if anæsthesia of the larynx is also present, portions may enter it. The extent and degree of the neurosis may be estimated by use of the probe. Occasionally anæsthesia is associated with painful sensations—anæsthesia dolorosa.

The treatment will depend upon the cause. When there is danger of the food entering the larynx the patient should be fed by means of an œsophageal tube passed below the cricoid cartilage.

*Hyperæsthesia*.—Abnormal sensitiveness of the pharynx is commonly associated with tuberculosis, gout, hysteria, alcoholism, elongated uvula, and conditions producing local congestion. The hyperæsthesia reveals itself when manipulations are carried out in the throat, and by the readiness with which slight irritation in this region excites reflex disturbances, *e.g.* coughing, retching.

No treatment is called for unless the condition prove a hindrance to the examination of other parts, *e.g.* larynx, naso-pharynx. Recourse should



then be had to cocaine, ice, and the education of the patient to tolerate the instruments.

*Paræsthesia*.—A great variety of abnormal sensations, unaccompanied by apparent pathological changes, may be experienced in the pharynx. Complaint may be made of pricking, smarting, rawness, dryness, heat, swelling, of the feeling of sand, a pin, hair, bone, or ball.

The subjects are usually neurotic, hypochondriacal, anæmic or dyspeptic, and women about the climacteric period, and those suffering from uterine affections, seem specially prone to this disorder. Frequently they imagine that the symptoms are due to cancer or phthisis. They are constantly examining their throats, and usually find "ulcers," "growths," or other non-existent alarming conditions. They describe their sensations with an air of self-conviction and elaboration of detail that bewilder one unacquainted with such ailments.

Before a diagnosis of paræsthesia is warranted all the possible sources of the abnormal sensations experienced must be considered. Thus, the less intelligent hospital patients frequently complain of soreness and burning in the throat when their symptoms are purely of gastric origin; recurrent attacks of "sore throat" unaccompanied by local manifestations may be found to be of a rheumatic nature; a feeling of irritation in an apparently normal pharynx is often associated with more or less nasal obstruction. All such conditions must be excluded.

It is also necessary to take into account the general health and temperament of the patient, *e.g.* anæmic and neurotic patients may complain of pharyngeal irritation which is traceable to a tiny granulation; of difficulty in swallowing, which is apparently due to a slightly enlarged lingual tonsil; or of soreness in the region from which a fish bone was removed days or weeks before: all of these conditions, experience has taught us, rarely cause symptoms in healthy individuals.

Keeping in view these facts, a thorough examination of the upper air tract must be made to discover, if possible, a local cause. In this search too much reliance must not be placed upon the patient's opinion as to the seat of origin of the symptoms, because sensations arising in the posterior wall of the pharynx may be referred to the region of the larynx, while others springing from the lateral parts of the pharynx may be felt in the corresponding ear. The tonsillar crypts and supra-tonsillar fossa should be examined for caseous collections, the lateral and posterior pharyngeal walls for granulations, the base of the tongue for enlarged lymphoid masses, as well as the naso-pharynx, nose, and larynx. The sensibility of the mucous membrane may be tested, but is found, as a rule, not heightened.

In the absence of any discoverable cause, a diagnosis of paræsthesia is justifiable. As our knowledge of disease advances, the class of paræsthesias will gradually be reduced; in the meantime, the more careful the consideration and examination of our cases, the fewer will it be necessary to relegate to this category.

An assurance that the ailment is not serious sometimes in itself causes no further heed to be paid to the symptoms. Usually, however, a prolonged course of treatment suitable to the patient's bodily and mental requirements is demanded. The affection may be very tedious, but ultimately the discomfort passes off or ceases to worry.

The use of sedative paints, sprays or lozenges, affords only temporary relief; the patient, however, may be dissatisfied if local measures are not employed. The galvano-cautery is sometimes useful for its psychical influence. Abnormal sensations caused by caseous tonsillitis, granular



pharyngitis, etc., are not true paræsthesias, but symptoms of these diseases which should be treated *secundum artem*.

NEURALGIA.—This affection is not often met with. The patient complains of severe remittent pain in the pharynx. Examination may fail to reveal a morbid process to account for the symptoms, or they may be traceable to an insignificant lesion. In either case the neuralgia is almost invariably the local expression of a general dyscrasia, and what has been stated in regard to the subjects of paræsthesia applies here.

The treatment should be mainly directed to the correction of the constitutional disturbance. If a pharyngeal affection is found associated with the symptoms, it should be treated according to its requirements; if not, local sedative applications may be tried, *e.g.* cocaine 5 to 10 per cent, but only for a short time, menthol 10 per cent, antipyrin 30 per cent, tincture of aconite, etc.

NEUROSES OF MOTION.—*Spasm*.—Tonic spasm of the pharyngeal muscles is commonly observed when the mucous membrane of the throat is suddenly irritated by the presence of something hard and rough in the food. Local inflammatory conditions and hysteria may be predisposing causes. In some individuals, especially neurotic females, the spasms may be readily excited and of frequent occurrence, and, coming on without warning, may cause the sudden ejection of food from the mouth, or retching until the source of irritation is removed. The condition is thus an annoyance to the patient and her friends. She may in consequence be compelled to masticate with undue care, to avoid certain articles of diet, and even to eat alone.

Clonic spasm of the palate, or pharyngeal nystagmus, is a rare affection in which, at varying intervals, the soft palate is rhythmically drawn upwards against the pharyngeal wall and then relaxed. This movement is due chiefly to the action of the levator palati, and causes a clicking sound. It may occur as often as a hundred and sixty times a minute (Oppenheim). Bond records a case in which the back of the pharynx moved rhythmically to the left, and the left side of the soft palate was drawn up. In a case of Seifert's there were in addition similar movements of the facial muscles. Spencer has seen nystagmus-like movements of the pharynx and larynx associated with nystagmus of the eyes. Lack places cases of this nature in two groups:—1. Those in which the soft palate is affected, the movements being apparently excited reflexly. 2. Those in which the pharyngeal and laryngeal movements are presumably due to severe nervous lesions.

The treatment of these spasmodic contractions resolves itself into the removal of all sources of irritation in the upper air tract, *e.g.* nasal obstruction, hypertrophic pharyngitis, etc. A condition as nearly normal as possible having been established, hygienic and dietetic measures, together with nervine tonics and sedatives, *e.g.* valerianates, or ammonio-citrate of iron with bromides, should be employed, while the patient exercises the volition to control the abnormal movements.

*Paralysis of the Soft Palate*.—Diseases involving the root or trunk of the spinal accessory may affect the innervation of the soft palate. When of central origin, neighbouring nuclei may be implicated, so that the palatal condition may be accompanied by defective movements in the tongue, larynx, sterno-mastoid, and trapezius. Paralysis of the palate alone is almost always due to diphtheria. Bosworth holds, however, that this paralysis does not necessarily follow a local affection, but may result from a general blood condition; while Garel has described an intermittent paresis which he attributes to neurasthenia.



The paralysis may be unilateral or bilateral. When complete, the symptoms are characteristic. The speech is nasal and open (rhinolalia aperta). Food is apt to pass into the nose, and liquids may flow through it. It is doubtful whether this symptom is not due to a concurrent paralysis of the superior constrictor.

The soft palate is found on examination to remain motionless during phonation and when probed. If the paralysis is unilateral the affected half hangs lower, the corresponding faucial arch is wider and less curved, the tip of the uvula may incline to the sound side, and on phonation the deficient movement of the paralysed half is strikingly apparent.

Paralysis of the constrictors is often associated with that of the soft palate. The former condition is recognised by the absence of wrinkling of the mucous membrane on one or both halves of the posterior wall of the pharynx during retching. Food tends to pass into the nose, and, if the lower pharynx is involved, becomes lodged about the epiglottis.

If due to a central lesion, the treatment will depend upon its nature. If diphtheritic, good results may be obtained by the local application of the Faradic current, together with strychnine, hypophosphites, or iron, internally.

### Acute Pharyngitis

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**DEFINITION.**—An acute inflammatory affection of the pharynx, in which the fauces, soft palate, and neighbouring regions are generally more or less involved.

**Ætiology.**—Four causes of acute pharyngitis may be recognised.

1. Idiopathic, resulting from exposure to cold, or sudden changes of temperature. As epidemics of the affection sometimes occur, associated with tonsillitis and constitutional symptoms, it is highly probable that a microbial element is the real exciting cause.

2. Toxic, “dependent on gouty or rheumatic conditions, or the action of various drugs, *e.g.* antimony, mercury, iodide of potash, and the sympathetic pharyngitis of various exanthemata, such as measles, smallpox, scarlatina, typhoid and typhus fever” (W. Williams). To these may be added the acute pharyngitis seen as an early syphilitic manifestation.

3. Septic, including erysipelas of the pharynx, “hospital throat,” acute phlegmon, etc.

4. Traumatic, resulting from injury from a foreign body, scalds, and inhalation of irritating vapours.

Among important predisposing causes must be included chronic nasal obstruction, the rheumatic and gouty diathesis, and sedentary occupations, especially when these are carried on in insanitary surroundings.

**Morbid Anatomy and Pathology.**—The mucous membrane of the pharynx and surrounding parts are at first swollen and hyperæmic, and the secretion is scanty and viscid. The degree of hyperæmia varies from a bright red in the idiopathic cases to the livid red met with in scarlet fever or erysipelas of the pharynx. The swelling of the inflamed tissues may be slight, or



so well marked that the uvula becomes enlarged to many times its natural size.

The tonsils are usually inflamed, and may be punctated with the yellowish accumulations characteristic of lacunar tonsillitis.

The inflammation generally tends to spread to the larynx, less commonly to the naso-pharynx and nasal cavities.

Acute pharyngitis may be secondary to acute rhinitis, resulting from simple extension of the catarrh by means of the naso-pharyngeal mucous membrane.

*Symptoms.*—The milder cases rarely come under medical treatment. In those of greater severity the symptoms are both general and local. Among the former are malaise, feelings of chilliness, and general aching of the limbs and muscles. The temperature may be raised to 100° or 101°, the tongue furred, and bowels constipated.

Locally, the throat feels hot, dry, and sore, and there is a frequent desire to swallow. If the uvula be much swollen, an irritating cough, and the feeling as if a foreign body was present in the throat, may be complained of. On inspection, the pharynx, fauces, tonsils, and uvula are seen to be red and swollen, and covered by a viscid secretion, which becomes freer some twelve hours or so after the commencement of the inflammation.

If the latter spreads to the larynx and trachea, there will be some hoarseness, and possibly a troublesome cough. A few days later a mucopurulent expectoration may indicate the extension of the catarrh to the larger bronchial tubes.

When the naso-pharynx is affected there will be greatly increased secretion, possibly slight deafness, earache, or tinnitus, from involvement of the Eustachian tubes. Symptoms of nasal catarrh (*vide* "Acute Rhinitis") will characterise the spread of inflammation to the nasal cavities.

In certain of the acute exanthemata the constitutional symptoms soon overshadow the throat lesions; but the latter are of importance, because they may be among the earliest manifestations of the graver malady. Moreover, in scarlet fever the pharyngeal inflammation may form one of the most serious complications of the disease.

In acute traumatic pharyngitis the inflammation is at first mainly limited to the site of injury. From thence, however, it may rapidly spread, especially if septic organisms gain access to the tissues through a broken surface.

The septic varieties of acute pharyngitis will, on account of their importance, be dealt with separately.

*Prognosis.*—In simple cases this is nearly always good; but a guarded prognosis should always be given in the case of children, because pharyngeal inflammation is often an early symptom of one of the acute exanthemata. In adults the possibility of the inflammation being an early syphilitic manifestation should always be borne in mind.

*Treatment.*—As a general rule it is well to give a saline aperient during the early stages of an attack. When this is severe the patient should remain indoors, in a well-ventilated room, at a temperature of about 65°. The diet should be light, while iced milk, milk and soda-water, lemonade, barley-water, etc., may be freely drunk. Sucking pellets of ice will often relieve local discomfort.

Medicinally, ten grains of "Dover's" powder, followed by a hot drink, is useful at the commencement of an attack, especially when the latter is associated with symptoms of acute nasal catarrh.

If rheumatic symptoms be present, salicylate of soda may be combined



with a diaphoretic mixture, *e.g.*  $\mathcal{R}$  sod. salicyl. gr. x., liq. ammon. acetat.  $\mathfrak{z}$ ij., spir. ætheris nitros  $\mathfrak{m}$ xx., aq. camph. ad  $\mathfrak{z}$ j. Sig.: to be taken every four hours until the pains are relieved. In this connection the guaiacum lozenges of the Throat Hospital Pharmacopœia are useful.

In cases where a gouty element is present free purgation, a light, unstimulating diet, followed by internal administration of alkalies and colchicum, will be appropriate treatment.

When the inflammation spreads to the larynx and trachea the following inhalation will give relief to the cough and local discomfort:  $\mathcal{R}$  Ol. eucalyp., ol. pini sylvest. ad  $\mathfrak{z}$ j., mag. carb. lev.  $\mathfrak{z}$ j., aq. camph. concentr.  $\mathfrak{z}$ j., aq. ad  $\mathfrak{z}$ iv. Sig.: The vapour arising from half-a-pint of water at 150° F., to which  $\mathfrak{z}$ ss. of the above mixture has been added, to be inhaled every two hours.

This may be accompanied by hot fomentations over the upper part of the windpipe and chest.

The sucking of pastilles containing borax, chlorate of potash, and cocaine will assist in removing the viscid mucus from the pharynx, and at the same time relieve some of the local discomfort. Œdema of the uvula, if slight, requires no special treatment; when it is excessive much relief may be afforded by making a few punctures with a fine scalpel, followed by the frequent use of warm alkaline gargle.

When the acute attack has subsided, the administration of quinine and iron, the hypophosphites or other tonics, and the local use of an astringent gargle, *e.g.* Borax grs. xxiv., glycerine  $\mathfrak{m}$ xx., tr. myrrh  $\mathfrak{m}$ xxiv., water to  $\mathfrak{z}$ j.; or even a daily gargle of salt water,  $\mathfrak{z}$ ss. to half a pint, will do much to hasten convalescence and promote a healthy condition of the pharyngeal mucous membrane.

**B. ACUTE SEPTIC INFLAMMATIONS OF THE PHARYNX.**—From clinical and bacteriological evidence Semon (and in this he was ably supported by the late Professor Kanthack) regards the various forms of acute septic inflammation of the throat, *e.g.* "hospital sore throat," acute œdema of the larynx and pharynx, phlegmon and erysipelas of these parts, and submaxillary cellulitis (Ludwig's angina), as identical pathological processes, varying only in their degree of virulence. While the streptococcus pyogenes is the organism most often associated with these acute septic inflammations, clinical evidence has been adduced to show that identical pathological conditions may occasionally be produced by the staphylococcus pyogenes, micrococcus tenius, and various forms of bacilli.

Acute septic inflammation of the throat may be primary or secondary. In the former it is probable that the organisms gain access to the tissues through some breach of the surface epithelium of the pharynx, fauces, or tonsils. In the second category the disease may spread inwards from the face or nose, and possibly arise in some cases from the pharyngeal tonsil.

In yet other instances the acute inflammation may start beneath the deep cervical fascia of the submaxillary region (Ludwig's angina), and secondarily infect the pharynx and larynx.

In such a case the primary inflammation may arise from cold; it is also said to occur in epidemics, as an extension from the inflamed glands of scarlet fever, or the poison may gain access by means of a wound or through a carious tooth. The disease is most common in alcoholics.

*Pathology.*—The inflammation of the submucous tissue varies much in intensity in the different forms of acute septic inflammation of the throat, and the resulting exudation may be serous, purulent, or even gangrenous.

While the inflammation may remain limited to the pharynx, it usually tends to spread to the larynx, and less commonly to the naso-pharynx.



The relation between such inflammations and the associated micro-organisms has already been referred to, but it may be pointed out that, as a general rule, streptococcal infection is more acute and virulent than that produced by the other pathogenetic organisms mentioned above.

*Symptoms.*—In the milder forms of septic pharyngitis, beyond the local discomfort, slight fever, headache, and constitutional depression, there is little to cause anxiety.

In the severer forms the disease may be ushered in by chills or rigors, a high temperature ( $102^{\circ}$  to  $106^{\circ}$ ), and a general feeling of malaise.

The pulse, at first full and strong, soon becomes more frequent and feeble, while the general prostration of the patient affords a good index of the evil effects of septic absorption upon the system. At the onset of the disease the temperature is usually high, but it then follows an atypical course, varying with the virulence of the poison and the degree of its absorption. The urine frequently contains sugar, and albumen is occasionally present. In all forms of the disease pain in the throat and dysphagia are early and well-marked symptoms. These are soon followed by a weak voice and difficulty of breathing, owing to the swelling of the pharyngeal and neighbouring mucous membranes. The fauces, uvula, and pharyngeal mucous membranes are deeply congested and swollen; the tonsils are red, swollen, and acutely inflamed.

The submaxillary cervical glands are always swollen and tender. As a rule the inflammation does not long remain localised to the pharynx, but passes downwards to the larynx, inducing, in addition to the symptoms already enumerated, a croupy cough, marked dyspnoea, and pain in and around the larynx.

If the tongue be firmly depressed, or a laryngoscopic examination be made, the epiglottis, aryepiglottic folds, and the arytenoid region will be seen to be swollen, red, and oedematous. This swelling may increase with such rapidity, and the dyspnoea become so urgent, that the patient's life may at any moment be in imminent peril from asphyxia. The dyspnoea is mainly inspiratory, because the inflammatory swelling is limited to the mucous membranes situated above the level of the vocal cords.

Such cases may, however, recover, especially when suppuration does not occur; the advent of the latter always renders the disease a very serious one, and especially so in those instances where the suppurative process is limited to the pharynx (Senator's phlegmon).

Under such circumstances a gangrenous slough may form in the sub-mucous tissues, and severe toxæmia rapidly supervene.

Sometimes the inflammation, serous or suppurative, spreads beyond the larynx to the tissues of the neck, inducing much swelling thereof, while evidences of pneumonia, pericarditis, pleurisy, peritonitis, or meningitis would indicate a more general infection of the system by the locally generated poison.

*Diagnosis.*—The sudden onset of the disease, the rapid spread of the inflammation, livid redness of the pharyngeal mucous membrane, lowered vitality of the patient, tendency to dyspnoea, swelling of the cervical tissues, and the frequent presence of sugar in the urine, or albuminuria, are symptoms which leave little doubt as to the real nature of the disease. The discovery of the *streptococcus erysipelatosus* in the inflamed areas will place the diagnosis beyond doubt (*vide* "Morbid Anatomy").

*Prognosis.*—This is always serious, except in the mildest cases, *e.g.* hospital sore throat. Death may unexpectedly occur, not only from asphyxia, but from cardiac failure, or less commonly from pulmonary,



meningeal, or other complications. Generally speaking, in a patient who is going to recover, the inflammatory symptoms quickly reach their height and rapidly subside.

*Treatment.*—The patient is of course confined to bed; the bowels should be opened by a castor-oil or saline aperient, an ice-collar applied to the neck, and he should have pellets of ice to suck. Every effort must be made to support the system, and to this end liquid nourishment, such as beef-tea, beaten-up eggs, and milk, should be administered hourly. If swallowing be painful, nutrient enemata should supplement food taken by the mouth. Cardiac depression must be combated by free administration of brandy, iced champagne, and full doses of strychnia. Semon speaks highly of oxygen inhalations in this connection. Judging from the favourable results following the use of anti-streptococci serum in cutaneous erysipelas, it should be tried in the disease under consideration; 10 cc. should be injected with the same precautions as are taken with the anti-diphtheritic serum, and the dose may be repeated in twenty-four hours if necessary.

Medicinally, the tincture of the perchloride of iron in full doses, combined with quinine, should be given every three or four hours. Salicylate of ammonium, in 20-grain doses every three hours, is especially useful when a high temperature is associated with delirium or cerebral complications.

Sprays of cocaine (15 per cent) to the pharynx and larynx will somewhat relieve the pain and congestion, and will aid in preventing spasm of the larynx.

Urgent dyspnoea which does not yield immediately to the application of 20 per cent cocaine must be dealt with by free scarification with a laryngeal lancet or a curved bistoury, shielded to within an inch of its point. Energetic counter-irritation by means of sinapisms applied to the throat, chest, back, and shoulder-blades should be tried, since its adoption has certainly seemed successful in reducing the pharyngeal inflammation in some cases.

Failing with these measures, laryngotomy or tracheotomy is imperative, and should not be delayed until the patient is semi-asphyxiated or *in extremis*. Intubation would be useless in most cases, because of the swelling of the pharyngeal and epiglottic mucous membranes above the larynx.

For the treatment of angina Ludovici, which may lead to pharyngeal and laryngeal symptoms identical with those above described, see article "Cellulitis."

C. HERPES OF THE PHARYNX.—This is met with as an eruption of small vesicles upon the pharynx and neighbouring parts, especially the fauces and soft palate. The pharyngeal mucosa are usually swollen and hyperæmic, and the small vesicles soon burst, leaving minute ulcers with a yellowish floor, surrounded by a bright red, well-defined margin. The onset of the disease is characterised by febrile symptoms and malaise, while the throat is sore and there is great pain on swallowing.

The treatment should be that laid down for cases of acute idiopathic pharyngitis. An iced spray of one-third per cent watery solution of ichthyol every quarter of an hour will quickly reduce the inflammation in this as in many other forms of acute laryngeal and pharyngeal inflammations.

LITERATURE.—WILLIAMS. *Diseases of the Upper Respiratory Tract*, 1901.—SEMON. See Paper read before Roy. Medico-Chirurgical Society, 23rd April 1895.



### Chronic Pharyngitis

**DEFINITION.**—A chronic morbid process resulting in hypertrophy or atrophy, and involving in a greater or less degree all structures in the mucous membrane lining the naso- and oro-pharynx, the process being pathologically and clinically distinct from that of acute pharyngitis.

For clinical convenience chronic pharyngitis is spoken of as *Simple chronic catarrhal pharyngitis*, *Hypertrophic pharyngitis* or *Granular pharyngitis*, *Pharyngitis sicca* or *Atrophic pharyngitis*. The incidence of the disease may be upon the sides of the pharynx, *pharyngitis lateralis*, and may extend to the Eustachian tubes. The uvula and soft palate, as well as the anterior and posterior pillars of the fauces, and the tonsils are not uncommonly also affected. From a pathological standpoint, as will presently be explained, these varieties really represent only different phases of one and the same disease; for clinical purposes the terms in vogue may usefully be retained so long as they do not lead to confusion.

**Etiology.**—It is a disputed point whether chronic pharyngitis is to be regarded as the result of one or more attacks of acute inflammation of this region. From a study of the clinical histories of a large number of cases, I am inclined to think that the morbid process is not an acute inflammatory one at the first, but that the process is insidious in its onset and progressive in its character. Its course is usually marked by repeated attacks of acute catarrhal sore throats, which are exacerbations of, and not the cause of chronic pharyngitis.

Moreover, a primary acute inflammatory condition of the pharynx, apart from the manifestations of the exanthemata, such as measles, scarlet fever, smallpox, typhoid, and typhus fever as well as syphilis, and also apart from catarrhal conditions occasioned by traumatic and toxic influences, is a very rare condition, and, therefore, can hardly be held responsible for the lesions in this region grouped under the term "Chronic Pharyngitis," which are of such common occurrence.

Although it is not the intention in this article to discuss acute pharyngitis, it seems necessary to the writer to make this preliminary reference to it, inasmuch as the acute and chronic condition has come to be, in his opinion, erroneously placed in a causal relationship, whereas, in a very large majority of cases, acute pharyngitis is merely a lighting up of a chronic morbid process.

In order to understand the etiology of chronic pharyngitis with a view to the treatment of the affection, it is important to bear in mind that although the oro-pharynx cannot be readily separated from the naso-pharynx, the former is really part of the alimentary canal, and the latter part of the respiratory tract. Speaking generally, when the stress of the affection is mostly upon the oro-pharynx, it is not uncommonly a manifestation of gastric disturbance, whereas, if the upper part of the pharynx is mainly involved, a cause may be found in the nasal cavities; at times the condition may be an extension by continuity from disease of neighbouring structures, such as the tonsils.

Indiscretions in diet, excessive use of alcohol and tobacco, and of irritating articles of food, such as condiments, must be mentioned amongst the foremost causes of chronic pharyngitis. At the same time, it would be erroneous to suppose that the pharyngeal mucous membrane becomes affected by the direct application of alcohol and irritating articles of food; the history usually points to a gastritis existing before the pharyngitis is developed, the latter being really evidence of the former. So also in the case of excessive tobacco-smoking, it is probable that the pharyngitis is secondary to the absorption of nicotine, and is subsequently aggravated by a continuance of the excess.

Constipation, cardiac disease, general excesses, and all conditions giving rise to venous congestion, are predisposing causes of chronic pharyngitis.

When the disease affects the upper pharynx, it is not uncommonly associated with a chronic naso-pharyngeal catarrh, and is indirectly due to some form of obstruction in the nasal passages. The extension of the process to the lower pharynx is probably due quite as much to the constant hawking by the patient, in his endeavour to get rid of an accumulation of mucus or muco-pus, which owing to its tenacious character is dislodged with difficulty, as it is to secretion tracking over the mucous membrane.



In pharyngitis sicca the atrophied condition of the pharyngeal mucosa probably has a cause in common with a similar condition met with in the nose and with which it is often associated. The dried and adherent secretion acts directly as a cause in the localised pharyngitis.

Any form of nasal obstruction, which necessitates mouth breathing, is indirectly a factor in chronic pharyngitis, occurring in persons exposed to an atmosphere charged with dust and irritating particles.

Dental caries of an extensive and active nature is so commonly met with in examining throats that it is difficult to assess its importance in the etiology of chronic pharyngitis. When the two have been associated to a marked degree, nasal obstruction with mouth breathing has also been present, and I am inclined to look upon the latter as a causation factor common to the two.

Excessive use of the voice is commonly stated to be one of the causes of this disease, and is put forward as an explanation of the hypertrophic variety met with amongst professional voice users. It is difficult to accept this explanation; the paralytic state of the pharyngeal muscles resulting from a pharyngitis already established, impairs the use of the pharynx as a resonance chamber, and in order that the walls of the pharynx may be adjusted to carry the voice to a distance a strain is necessitated. It would, therefore, seem that, on the contrary, chronic pharyngitis may be a cause of excessive use of the voice, and that the latter does not produce but only aggravates the former.

Improper use of the voice, on the other hand, may lead to chronic changes in the walls of the pharynx. But in estimating or treating faulty voice production as a cause, we must not overlook the fact that professional voice users are exposed to vitiated atmospheres, late hours, and irregularities in diet, and that physiological transgressions are often as much responsible as transgressions of natural limits of normal registers of voice.

Certain diatheses, such as that of scrofula, anæmia, tuberculosis, syphilis, are usually mentioned in discussing the etiology; I am inclined to think that these causes, as well as the arthritic diatheses, play a less important part in the disease than is commonly believed.

Diabetes and myxœdema have both been brought under my notice by the pharyngeal conditions complicating these diseases. The most marked case of pharyngitis sicca I have seen occurred in a diabetic who sought relief solely for the disease of the throat. The atrophied and dried condition of the mucous membrane completely involved the entire pharynx, and extended over the mucous membrane of the posterior third of the larynx. In pharyngitis sicca of an extensive nature it is as well to examine the urine for sugar.

In myxœdema the mucous membrane of the pharynx and adjacent soft parts may become thickened and œdematous, and give rise to symptoms of chronic pharyngitis which bring the patient to the throat clinic.

*Pathology.*—In sections taken from the less advanced cases, the prominent change met with is a proliferation and dilation of the blood-vessels beneath the mucous membrane; the hyperæmia is distinct from that met with in acute pharyngitis, inasmuch as it is less intense and less diffuse. Owing to this local hypernutrition an increase of the lymphadenoid tissue of the mucosa takes place, and thickening results. A hyperplasia of the connective tissue also occurs, and is a factor in the increase of the mucous membrane, more particularly about the pillars and recesses of the fauces, and the lateral wall of the pharynx, giving rise to the condition known as pharyngitis lateralis. A more or less dense small cell proliferation accompanies this form of hyperplastic fibrous tissue, and may extend into the deeper layer amongst the muscle fibres, and so be a factor in the impaired mobility and accommodation of the pharyngeal muscles for vocal purposes. In other sections from the same pharynx atrophic patches are met with in which the lymphadenoid tissue is reduced in amount, and the connective tissue is also atrophied.

Various changes are met with in the epithelium. This in places may be thinned, or even ulcerated over congregations of lymphadenoid tissue, and in other places it may be thickened, giving rise to white patches. The follicles may undergo caseous degeneration (follicular pharyngitis) and give rise to minute follicular ulceration.

*General Symptomatology.*—It is interesting to note that in the larynx gross changes, such as paralysis of a cord or a new growth, may develop and exist for some time without giving rise to any symptoms, and perhaps only accidentally be detected; whereas a comparatively slight lesion in the



pharynx will cause a patient to seek relief at a time, perhaps, when the departure from the normal may be so slight as to escape notice at a superficial examination.

The symptom usually complained of is discomfort, or an unpleasant sensation in the throat. This is variously described as a sense of a foreign substance necessitating a constant clearing, or inability to clear the throat, and irritating cough. At times the discomfort is spoken of as an aching or dragging sensation, which may be referred to a level as low as the interclavicular notch. The discomfort seldom amounts to pain, but pain of a shooting character passing up to the ears may be complained of. In the hypertrophic form, with much secretion of mucus, a soreness in the throat is experienced; if the atrophic condition predominates a dryness is mentioned. These symptoms are usually more marked on rising and after prolonged use of the voice. At times a sense of impending suffocation in sleep may be the cause of treatment being sought.

The subjects of chronic pharyngitis have not uncommonly experienced symptoms for some time when they come under observation. The one symptom above all others which decides the sufferer to seek relief is impairment of vocal power, it may be hoarseness, or it may not amount to more than weakness or uncertainty of voice. During the more acute exacerbations dysphagia may be present.

*The Objective Evidence and Diagnosis.*—Too much stress cannot be laid upon the necessity of examining the buccal cavity thoroughly, and with a good reflected light, in order that a precise knowledge may be obtained of the condition giving rise to the distressing symptoms, and the cause removed.

In the chronic catarrhal and hypertrophic variety the tongue is usually coated and dirty, the uvula may be swollen and elongated, the adjacent parts of the soft palate and fauces congested, the tonsils inflamed and oedematous, the patient most intolerant to examination, and readily retching at the approach of a tongue depressor.

In a less acute stage the naked-eye evidences of morbid action in the mucous membrane lining the naso- and oro-pharynx may be so slightly marked, or so hidden, as at first glance to escape observation. The mucous membrane may be found to be reddened and studded with nodular excrescences of about the size of a pin's head, and not exceeding that of a split mustard-seed. They may be so closely set as to give a papillomatous appearance, or the mucous membrane may be unevenly hypertrophied, and thrown up into irregular ridges. The surface may be unduly moist, owing to excessive secretion, and covered with a film of saliva and minute air bubbles, or it may be unnaturally dry. In the latter case the membrane is thinned, smooth, and glazed, having a parchment appearance, and constitutes the condition of atrophic pharyngitis, or pharyngitis sicca. In this form nasal secretion can often be seen dry and adherent in the upper parts. This atrophic condition in one part may be associated with a hypertrophic condition in another, so that the classification of the various forms of chronic pharyngitis usually described breaks down. The tonsils commonly share in the morbid process, and should the patient retch in the course of the examination, the lateral walls of the pharynx will be brought into view, and may present as red thickened bands (pharyngitis lateralis).

It has already been said that the naked-eye evidences of morbid changes may be so slightly marked as to escape detection; the departures from the normal may at times appear altogether insufficient to account for the discomfort complained of. At other times a pharynx giving rise to no



discomfort, but examined in the course of the investigation of some aural or nasal condition, may present marked appearances of a chronic hypertrophic or glandular pharyngitis. In connection with this mention may be made of the granules seen in the pharynx in children. In them the etiological factors, alcohol, tobacco, vocal fatigue are absent; the presence of little projecting granules, varying in size from a pin's head to a pea, cannot be regarded as due to any morbid process, and does not give rise to any symptoms. These granules are part of a tendency to lymphatic enlargement, and are often associated with adenoids.

When the voice is affected an examination of the larynx in the majority of cases will probably be unproductive of any lesion being found, excepting perhaps some minor degree of thickening of the mucosa in the inter-arytænid space. The explanation I have to offer of the vocal impairment in the absence of any laryngeal lesion is that given in discussing the etiology of the disease. On theoretical grounds it is conceivable that the impairment of the muscular action of the pharynx, which is directly due to the morbid process, may by reflex action weaken the laryngeal muscles for the freer manipulation of tones of the singing voice. On the other hand, it must not be forgotten that the subject of chronic pharyngitis suffers most when the general health is run down, and the vocal weakness may be quite as much evidence of general loss of tone as of the pharyngitis itself.

Without attaching undue importance to lesions in the post-nasal space and the nasal cavities themselves, it may be stated that no examination of a case of chronic pharyngitis can be considered to be complete unless these upper air-passages have been carefully inspected.

Naso-pharyngeal catarrh, hypertrophic rhinitis, and obstructive nasal lesions, when present, are usually more than merely complicating disorders; they stand in direct causal relationship to the pharyngeal disease, and their removal is necessary to eradicate it.

*Treatment.*—There is, perhaps, no disease of such frequent occurrence as chronic pharyngitis, which occasions the patient by its persistency so much distress, and the practitioner by its rebelliousness to treatment so much disappointment, a disappointment which is only aggravated by the trivial importance of the disease, trivial only so far as more grave results are not likely to ensue. And yet the treatment is simple, and success can generally be looked for, provided a diagnosis of the situation and cause of the disease is arrived at, and the patient possesses the moderate intelligence and strength of will required to co-operate in the treatment. The lack of success, I think, in many cases must be attributed to insufficient illumination being used in the examination of the parts, all of which can be seen by direct inspection, and to insufficient attention to detail in diagnosis and treatment.

It follows from what has been said in discussing the etiology that when the pharyngeal disease is a secondary affection, as it so frequently is, the treatment of the general health is of first importance. Should the disease be attendant upon dyspepsia or gastritis, errors in diet must be amended. Into the general rules of diet and treatment of gastritis we need not enter in this article, but the importance of relieving any plethora or constipation that may be present cannot be too frequently dwelt upon. A teaspoonful of some aperient salt, such as Carlsbad, may be taken in a tumbler of hot water on rising, or if the bowels are more constipated the following pill may be prescribed: Pil. rhei co., pil. colocynth c. hyoscyamo, āā gr. iiss., one or two at night.



But even when the pharyngitis is but a manifestation of some general disorder, local treatment is often required. Speaking generally, gargles are of but little service; they seldom reach the affected parts, excepting in the acute stages of a chronic catarrhal pharyngitis, when the tonsils, uvula, and soft palate are inflamed. In this condition a gargle containing chlorate of potash or alum  $\mathfrak{z}$ iss., tincture of myrrh  $\mathfrak{z}$ ij. to  $\mathfrak{z}$ viiij. may be usefully ordered. As the acute stage subsides astringent pigments, such as nitrate of silver or sulphate of zinc, ten to twenty grains to the ounce, may be applied at intervals of two or three days. Lozenges are so commonly taken that beneficial effects are naturally attributed to them; but when a gastric disorder has to be combated it is as well to be sparing in the administration of astringent drugs by the mouth. Pastilles are decidedly preferable to the hard lozenge, with its rough and angular edges. All the drugs that are known to be of benefit in relieving pharyngitis can now be obtained in the form of pastilles. One containing chlorate of potash gr. ij., borax gr. j., and cocaine one-twentieth of a grain, and another containing menthol and cocaine, a twentieth of a grain of each, in my experience, have afforded considerable relief.

When the uvula is so elongated as to be obviously a source of irritation, it can easily be reduced. Its removal may be followed, however, by a sharp reaction, and little or no benefit. An application of a 10 per cent solution of eucaine will give sufficient anæsthesia to permit of the tip being held and drawn downwards by a pair of forceps and cut through with curved scissors, care being taken to leave the cut surface on the posterior aspect.

If the pharyngitis is kept up by any obstructive lesion in the nose this must be removed. A nasal or post-nasal discharge may be treated by syringing through the nostrils a solution containing a teaspoonful of the following powder, pulv. sodæ bicarb., pulv. sodæ bibor.,  $\mathfrak{a}\mathfrak{a}$   $\mathfrak{z}$ iiij., pulv. sacchar. alb.  $\mathfrak{z}$ iiij., to half a tumbler of warm water.

A spray consisting of menthol gr. xx., eucalyptol  $\mathfrak{m}$ xl., in an ounce of paroleine, may be ordered for the nose and naso-pharynx, and is useful when the pharyngitis is part of a post-nasal catarrh.

When the tonsils, or the lateral folds, or the lymph follicles are so enlarged as to materially assist in the persistency of the pharyngitis, and other remedies have been found inefficient, then these structures must be dealt with by more active measures. A diseased tonsil, without being enlarged, and even when hidden between the pillars of the fauces, may play a very important part in a chronic pharyngitis, and is very liable to be overlooked as an exciting cause. Upon drawing back the anterior pillar a shrunk tonsil may be found inflamed and its follicles plugged, the treatment of which, either by enucleation or the galvano-cautery, has in my experience resulted in a cure of a pharyngitis which has resisted every other mode of treatment. When a tonsil is materially enlarged it can, of course, be removed by the guillotine, but the wisdom of doing so whilst it is acutely inflamed is doubtful. The enlarged lateral folds can be reduced by the cautery; some have advocated excision with the knife.

Although it may be a disputed point whether the hypertrophied tissues and lymph follicles on the posterior wall of the pharynx can account for the pain and suffering that at times accompany their presence, there can be no difference of opinion amongst those experienced in the treatment of granular pharyngitis about the relief afforded by the destruction of these hypertrophied follicles. The treatment of granular or hypertrophic pharyngitis consists essentially in the destruction of these hypertrophied



structures. This is most effectively done with the galvano-cautery. The surface of the pharynx is wiped dry, and a 10 per cent solution of cocaine or eucaine is applied with a pledget of cotton-wool to the particular follicles to be attacked. It is better not to cauterise too many follicles in close proximity; it is also as well to keep as far as possible to one side of the pharyngeal wall at one sitting.

A small round cautery point is heated to a dull red and plunged in the centre of the follicle. A white slough forms and falls off in about a week, when other follicles may be dealt with.

In pharyngitis sicca, or atrophic pharyngitis, a good deal can be done for the patient's comfort by the removal of the accumulated dried adherent mucus in the naso-pharynx and the nose itself. This can be effected by an alkaline nasal douche and throat spray. The recurrence of these accumulations I have found to be checked, and the mucous membrane of the pharynx improved by being somewhat vigorously rubbed with swabs of cotton-wool soaked in a solution containing iodine gr. iij., iodide of potassium gr. xv., carbolic acid gr. iij., glycerine ʒj., water to ʒj. The strength of this solution may be increased.

There is one important point in the treatment of pharyngitis which is more liable than another to escape attention, and that is the necessity for obtaining for the pharynx as much rest as possible, not only in keeping from it irritating articles of food, and interdicting alcohol and tobacco, and enjoining fresh air and the avoidance of foul atmospheres, but more especially in impressing upon the patient the necessity of forgetting his throat. That constant effort to clear the throat, to get rid of an imaginary foreign body, or to remove mucus or saliva, necessitates a continuous working of the pharyngeal muscles, and explains the aching and dragging sensations referred to the throat and lower part of the neck. Many subjects of chronic pharyngitis are also mouth-breathers by night, if not by day, so that the mucous membrane is irritated continuously in one way or another day and night. Rest is most essential for healing, and rest for this region is most difficult to obtain.

### Chronic Infective Diseases

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**SYPHILIS.**—Of the chronic infectious diseases syphilis is the one most prone to attack the fauces and pharynx, and at all stages of the disease. It may present as a primary chancre, as an erythema of the fauces, as mucous patches, as superficial ulceration, as gummatous deposits followed by deep ulceration, or it may leave evidence of its past activity in the form of cicatrices, destruction, and deformity.

Primary infection of the fauces is of rare occurrence; cases when it occurs are usually recorded, but the precise manner of infection is not always easy to ascertain. The tonsils, especially when hypertrophied, have commonly been the site of infection. The chancre here usually covers a larger area than on the penis, the corresponding lymphatic glands become indurated. The condition is liable to be confused with epithelioma; treatment and the appearance of a secondary rash will clear up the diagnosis. Erythema of the fauces is a common manifestation of the secondary stage, and gives rise to so little inconvenience that it commonly escapes notice until the throat is examined in making a diagnosis of the cutaneous erup-



tion which appears about the same time. The soft palate along its free border and the pillars of the fauces are the parts attacked, and a fairly sharp line of demarcation serves to distinguish the erythema from similar eruptions.

Mucous patches usually appear within six weeks to three months of the infection, but they may appear at any time in the course of the disease. The patches are but slightly raised, and present a grayish white or mauve appearance along the margins of the soft palate, uvula, and pillars of the fauces, and the surface of the tonsils, but are not commonly met with on the pharyngeal wall.

Superficial ulceration is generally placed amongst the secondary manifestations of the disease. It is difficult to lay down definite characteristics of these ulcers. They may form a later stage of a mucous patch or of a small gumma. They are commonly oval, elongated with a dirty sloughy base, and have a tendency to a serpiginous arrangement. They may have to be diagnosed from tuberculous ulceration which is rare. Occasionally one sees in the upper part of the tonsil a condition simulating a specific oval ulcer, which is not an ulcer at all, the appearance being due to the deposit of muco-pus from a follicle in one of the furrows which commonly exist in the upper part of the tonsil. This condition has been described as following an attack of tonsillitis, and is liable to be mistaken as evidence of specific disease.

Gummatous tumours must be regarded as one of the rarer manifestations of the disease. A distinct tumour may present on the hard palate, or on the pharyngeal wall, more commonly it has broken down, and resulted in deep ulceration when it comes under treatment.

Deep ulceration may occur in any part of the fauces and pharynx, and the appearances are too well known to need further description. It must not be forgotten that deep destructive ulceration, the result of hereditary syphilis, may occur early in life. Mention must also be made of congenital gaps and malformations in the structures of the fauces, which at times have been erroneously regarded as evidence of past specific disease.

The treatment of syphilis when it affects these regions is essentially the same as when other regions are attacked.

*Tuberculosis.*—Having regard to the frequency with which tuberculosis is met with in the larynx, the manifestation of the disease in the fauces and pharynx may undoubtedly be spoken of as of rare occurrence. There is not sufficient data to prove that the region we are considering is ever the site of a primary infection. In the cases recorded it has generally been associated with the disease in the lungs and larynx. When no signs pointing to pulmonary tuberculosis have existed at the time the disease was diagnosed in the pharynx, they have subsequently developed, so that it is reasonable to infer that deposition of tubercle had previously taken place in the lungs.

The onset of the disease is usually insidious, and if the larynx is not also materially affected the symptoms may not be sufficiently marked to bring the patient under observation until the stage of ulceration has been reached. Ulceration is attended with the usual symptoms of sore throat, and deglutition becomes difficult. Owing to the paresis of the soft palate resulting from the infiltration and ulceration, food and drink find their way into the nasal chambers. Symptoms and signs of the pulmonary disease are commonly also present, and the larynx may be so extensively involved as to overshadow the symptoms produced by the faucial disease.

A case in its earlier stages, before it is blurred by the results of ulcera-



tion, affords an exceptional opportunity for the clinical study of the development of tubercle. The velum, immediately adjacent to the base of the uvula, is usually the starting-point, and thence the disease spreads by continuity. The pathological process is essentially the same as when tubercle develops in the larynx; it commences in the submucosa and gradually spreads outwards. Small grayish white nodules appear projecting on the surface of the palate, which generally presents some degree of pallor. The nodules break down, leaving minute points of ulceration which coalesce and give the velum a peculiar mouse-nibbled appearance. The area affected increases and becomes coated with a sloughy membranous deposit, which upon being wiped away leaves a raw bleeding surface. The process may be rapidly destructive, and its characteristic features proportionately less distinct.

The diagnosis of the disease in its earlier stage may be confounded with that of diphtheria. In a case that came under my notice, the patient had been certified as suffering from diphtheria, and had been under observation in a fever hospital for several days, but the bacteriological examinations of the throat were negative of diphtheria. The clinical appearances of the fauces were, one need scarcely add, very suggestive of diphtheria. Scrapings from the palate, however, revealed tubercle bacilli, the lungs and larynx also gave evidence of tuberculosis. In the more advanced cases syphilis is perhaps the disease from which the diagnosis would have to be made. The points of difference are briefly these: in secondary syphilis the ulcers are elongated and oval, and tend to spread along the margin of the palate from immediately above the tonsils towards the uvula; in tuberculosis the ulcers are minute and circular, and commonly radiate outwards from the base of the uvula. In syphilis the palate is reddened, in tuberculosis pallid, and there is evidence of tubercle in other organs, the general health being also more markedly affected than in syphilis. The two diseases may, of course, be associated.

Treatment must be general as well as local. Into the general treatment one need not here enter; locally, orthoform can be insufflated to relieve the dysphagia and permit of sufficient nourishment being taken, it may be combined with resorcin, and is preferable to morphia and repeated applications of cocaine. In suitable cases the affected area may be curetted, and lactic acid in solutions of increasing strength rubbed in at intervals. When this is not practicable iodoform has been found of service.

*Lupus*.—When lupus attacks the fauces it is usually secondary to the disease in the integuments of the face and nose. The body of the uvula is commonly the starting-point, and hence it spreads along the soft palate to the pillars of the fauces.

The morbid process is essentially one of infiltration producing nodular thickening of the part affected. Ulceration follows, but the process is slowly destructive—cicatricial tissue developing side by side with the ulceration. This chronicity of the disease and the efforts of nature to arrest give the part a characteristic appearance, and assist to distinguish it from other infective processes which attack this region.

From tuberculosis it may be further distinguished by the general health not being affected. The ulcers and cicatricial tissues may be confounded with that produced by tertiary syphilis; in lupus the ulcers are not so deep, nor is the destruction of the adjacent structures so extensive. Moreover, if the patient is placed under antisyphilitic treatment, lesions due to lupus, instead of healing, would probably be aggravated. The diagnosis may have to be made from epithelioma; the diffuse infiltration of lupus would help



to distinguish it from malignant disease which partakes more of the nature of a tumour. Moreover, tuberculosis, syphilis, and malignant disease affecting the soft palate do not remain under observation for many days without giving evidence of the progressive character of these diseases; lupus, on the other hand, would remain comparatively stationary.

The treatment must be directed towards improving the general health by means of tonics and fresh air. Tuberculin may be used for therapeutic as well as diagnostic purposes, and without danger, provided it is properly injected. When localised, the diseased tissue may be cauterised or curetted, and subsequently treated with lactic acid as suggested by Krause.

*Leprosy.*—The opportunities for observing leprosy in the living subject in this country are but few. When it appears on the fauces, uvula, pharynx, or tongue, it is a secondary manifestation of the disease. The infiltration is followed by ulceration and adhesions which might be suggestive of syphilis were not the general disease by this stage well established in other parts. Towards the end the disease may be associated with tuberculosis.

*Rhinoscleroma.*—In this disease, to which reference was made in discussing chronic infectious diseases of the nose, the pharynx and nasopharynx are regions that are by no means infrequently involved. The disease is characterised by dense and hard infiltration with tendency to contraction and cicatrisation, but not to ulceration. The diagnosis is assisted by the fact that cases observed in this country have come from parts where the disease is endemic; should further evidence be required, the specific bacillus and also the cells of Mikulicz may be looked for.

**Retropharyngeal Abscess**

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*DEFINITION.*—A collection of pus situated in the connective tissue between the posterior pharyngeal wall and the cervical vertebræ.

*Etiology.*—The disease is essentially one of infancy, 75 per cent of the cases occurring during the first year of life, while it is very rare in children above four years. Retropharyngeal abscess is generally idiopathic, occurring in weakly, ill-nourished, or rickety children, or in those in whom an attack of one of the acute specifics has lowered the general vitality of the system. In these cases probably a septic infection of the nasal or pharyngeal mucous membranes has been the starting-point of disease. Less frequently the affection is secondary to cervical caries, suppurating tubercular cervical glands, suppurative middle ear lesion or its sequelæ, and still more rarely it may result from traumatism.

*Morbid Anatomy and Pathology.*—The idiopathic abscess is a suppurative lymphadenitis of one of the retropharyngeal glands of Henle, situated one on each side of the middle line opposite the second and third cervical vertebræ. These glands disappear between the third and fifth year of life, and hence the confinement of the disease to the early period of childhood. In the exceptional adult idiopathic cases suppuration occurs in the degenerate remains of the glands referred to. A retropharyngeal abscess is unilateral, and generally most prominent in the oro-pharyngeal region. Though usually circumscribed, the pus, if not evacuated spontaneously or artificially, may burrow in various directions, *e.g.* towards the larynx, laryngis, producing



œdema, towards the angle of the jaw, external auditory meatus, etc. The glands behind and below the angle of the jaw on the same side as the abscess are usually inflamed. In the secondary forms the morbid anatomy will vary with the nature of the primary lesion.

*Symptoms.*—In idiopathic cases, dysphagia and difficulty of swallowing are early symptoms, and may be accompanied by pyrexia with its constitutional symptoms.

These are soon followed by dyspnoea and a croupy cough, while the voice becomes aphonic, throaty, or hoarse. The head is held stiffly, and the glands behind and below the angle of the jaw are inflamed.

In secondary cases the symptoms may be much less acute, and pyrexia entirely absent.

Examination of the oro-pharynx in acute cases will reveal a prominent swelling of the pharyngeal wall which fluctuates on palpation.

*Diagnosis.*—In infants, the symptoms enumerated may at first suggest membranous laryngitis, but the difficulty of swallowing, bulging of the pharyngeal wall, unilaterally enlarged glands, and fixation of the head, should suffice to differentiate the diseases. In adults, a breaking-down gumma may closely simulate retropharyngeal abscess, but the exhibition of iodides would soon settle the diagnosis.

*Prognosis.*—This is usually good if treatment be undertaken early. Delay involves the risks of asphyxiation from œdema of the larynx, closure of the glottis by pressure of the abscess, bursting of the abscess during sleep, which may result in immediate asphyxia, or a subsequent attack of broncho-pneumonia. In secondary forms the prognosis necessarily depends upon the nature and gravity of the primary disease.

*Treatment.*—Once the diagnosis is made, the abscess should be evacuated without delay. Two methods, the external and internal, have been advocated, and both have given excellent results.

If the intra-oral method be chosen (and it has the merit of simplicity), the child should be lightly anæsthetised, and a vertical incision made through the prominence of the abscess, while the head hangs slightly backwards over the end of the table, in order to avoid the entry of pus into the larynx. The abscess having been evacuated, the patient should be immediately turned upon his side. If the abscess be large, it would be preferable to aspirate it before making the incision.

Marked laryngeal stridor may necessitate tracheotomy as a preliminary to evacuation of the abscess.

When the latter is secondary to caries of the spine, Chiene advised an external operation in order to avoid septic infection of the abscess cavity from the mouth. Pollard has adopted same method in the acute idiopathic abscesses of early childhood, and recommends a one-inch incision along the posterior border of the sterno-mastoid, commencing the same distance below the mastoid process.

The carotid vessels and vagus nerve are held well forwards, the abscess cavity opened, rendered as aseptic as possible, and a drainage tube inserted into its cavity.

Suitable constitutional treatment should be adopted in all forms of the disease.



**Pharyngocele.**—The term designates a diverticulum from the pharynx. Such a diverticulum or pouch is exceedingly uncommon. The orifice connecting it with the cavity of the pharynx is usually at the lowest part of the pharynx posteriorly, whilst the pouch lies behind the pharynx or œsophagus and is directed downwards backwards, and, as a rule, to the left side. Probably the most correct view as to the origin of such a pouch is that it is due to defective closure of a visceral cleft, this supposition being more satisfactory than the view according to which pharyngeal pouches are not congenital but acquired, whether by pressure from within or by traction from without, as œsophageal pouches are. A pharyngocele being, therefore, regarded as congenital has a definite relationship to branchial cysts (*vide* "Neck," vol. viii.) and congenital pharyngeal fistulæ. The pouch is at first of small size no doubt, but in the course of years becomes slowly though steadily enlarged by pressure of food within it, and the pouch as it enlarges gradually extends downwards. Its size varies greatly in different cases, and it is sometimes pedunculated (*vide* *Brit. Med. Jour.* 1901, I. 1204). Its wall is formed by layers representing the mucous and submucous membranes of the pharynx, whilst if the pouch is of small size there is usually a muscular layer also.

*Symptoms.*—The patient, in the great majority of cases, yet not invariably, is of the male sex, and it is seldom before middle life that the pouch has attained sufficient size to have been detected or to have caused inconvenience. When large enough, however, to do so, the chief symptoms are either interference with deglutition or such as are due to pressure on the trachea, blood-vessels, or recurrent laryngeal nerves. At the same time there is a soft, more or less tympanitic, swelling on the left side of the neck at its upper part, and this swelling can be emptied by pressure, when the contents will pass into the mouth or œsophagus. The condition is most likely to be mistaken for a stricture of the œsophagus, the possibility of which can be excluded by the passage of a bougie into the stomach, and only in the event of the instrument entering the pouch is there any difficulty in diagnosis. The only possible treatment is the surgical removal of the pouch.

**Phimosis.** *See* CIRCUMCISION.

**Phlebitis.** *See* VEINS.

**Phlegmasia Alba Dolens.** *See* PUERPERIUM.

**Phosphaturia.** *See* URINE.

**Phosphorus Poisoning.** *See* TOXICOLOGY and TRADE DISEASES.

**Phthisis.** *See* LUNGS.

**Physical Education.** *See* THERAPEUTICS.



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PHYSIOGNOMY AND EXPRESSION as aids to the diagnosis of disease have been dwelt upon by the older writers on medicine more than by those of recent times. Observers, when giving their descriptions, have always placed stress upon the facts they have seen themselves and regard as the most uniform occurrences characteristic of a certain disease or form of illness. The advance of scientific medicine, with the aid of the stethoscope, the clinical thermometer, the microscope, and chemical examination of the urine and secretions, as well as bacteriological investigations, have supplied more certain criteria for the diagnosis of disease than were formerly available. Still it remains true that many points in physiognomy indicate either proneness to disease or aptness in resisting power. This is specially seen in studying the diseases and illnesses of childhood.

It is necessary to distinguish observations and descriptions of physiognomy from those of expression. The older writers on physiognomy say very little about mobile expression, and the association of conditions of health or brain status with the types described. Lavater figured the cast of head and features which appeared to him indicative of phases of mental character, but he understood very little about expression. Sir Charles Bell (1) was one of the first inquirers to differentiate clearly between expression and physiognomy, showing that the former depends upon and directly indicates action in the nerve system.

Physiognomy deals with the form of visible parts of the body, the head, the separate features, and the palate. Form depends upon relations in size and in proportion; physiognomy is characterised by proportioning of the head and features and their parts in developmental growth as well as size. When these points are described in a patient, together with the conditions of health and brain associated, a valuable clinical account is presented of great use in acquiring experience. In studying physiognomy and anthropology instruments of precision may be used (3). It is, however, necessary, when observing the body, to follow methodical procedure, and to know what to look at and the characteristics to describe, so that observations may be rendered comparable and uniform; at the same time we should learn to distinguish normal from subnormal development (19). Expression differs from physiognomy, and varies with the changing action of the central nervous system, whose



states are indicated by the neuro-muscular system—just as the hands of a watch express the movements of the wheels inside. Expression in the face is the finest index of changes in the brain-centres as produced by the muscles acting on the cutaneous surface, and may vary as rapidly as the words uttered in expressing thoughts. Still, though the expression be evanescent, it is in many particulars capable of description, and is especially important in the clinical study of mental cases. Expression results from movements in other parts than the face, such as gesticulations and action of the hand and the attitudes of the head and body. Movement is the principal means by which we ascertain the condition and action of the nerve-centres; further, movement and results of movement are the only means by which mental action in another person becomes known to us (11-14). It is now clearly understood that expression is produced by movement and muscular tone, resulting from the balance of action in the brain-centres. Thus expression may be described as a direct sign of mental status, while physiognomy indicates prenatal and congenital development.

Physiognomy is here dealt with as an aid to clinical description and diagnosis, not as applied to ethnography. I think that physiognomical points should be recorded in the clinical description of a patient, as indicating a general condition of development, normal or subnormal. Descriptions of each point in bodily development—when the co-existing status of general nutrition and the expression of brain-action are simultaneously recorded as normal or subnormal—have added much to our knowledge of the pathology, etiology, and prognosis of clinical cases. The correlations of physiognomy and nutrition of body with the expression of brain-action afford most useful information and explanation of many problems. The fact that a physiognomy subnormal in certain particulars is much associated with a low status of nutrition adds interest to this subject, and though the relation may be empirical, the correlation as cause and effect appears to be established by experience. The study of physiognomy and expression as applied to medicine is based on observation. We need to recognise normal from subnormal development in our patients as seen in each feature, and in growth of the body as indicated by anthropometry (5).

Among indications of the developmental status of an individual the general conformation of the head and detailed points in physiognomy are of much interest. For the purposes of clinical medicine the volume or content of the brain-case is of more importance than the form; next to this the conditions of ossification and growth of the bones composing it have the greatest interest. The head and the brain grow rapidly in early life. In the infant at birth the head has an average circumference of 13·95 inches in the male and 13·75 in the female, increasing to about 17·5 inches at nine months and 19·0 inches at twelve months, continuing to grow until it becomes 20 or 21 inches in circumference at seven years of age. In form the general contour of the head is rounded; the forehead should be smooth, without frontal ridge or contraction between the temples. The greatest transverse diameter is between the centres of the parietal bones behind the ears, and at a higher level than their insertion. The anterior fontanelle enlarges with the growth of the head till about the eighth month, then becoming gradually closed by growth of the frontal and parietal bones, so that it is hardly to be felt after the first year.

The head should be viewed both full face and in profile. It may best be felt by placing the open hand on the head, with the thenar and hypothenar eminences resting on the forehead, so that the extended fingers, lying on the surface, may feel the general form and volume, as well as any



irregularities in ossification. Measurements may be taken with a tape at the largest circumference, transversely from one meatus to the other, over the top of the head, and again, antero-posteriorly in the median line from the root of the nose to the occipital protuberance. The size, the growth in a period of time, and to some extent the configuration or proportions of the head, are thus observed. The most important information to be gained is as to the size and content of the brain-case; next to this the conditions of ossification are of great interest, and these—if we exclude those due to rickets and syphilis as results of disease—are mainly states of ossification, either at the centres of the bones or near their margins, where the sutures may be thickened and raised, principally at the interfrontal and interparietal sutures. The frequency of deviations from the normal cranial physiognomy, as far as regards boys and girls of school age, is shown in the following quotation from the report (4) on 100,000 children I examined in conjunction with a committee, 1888-92.

Among 53,144 boys, 46,856 girls seen, there were reported as follows:—

*Cranium defective*, including all cases below, 2334 boys, 1659 girls.

*Cranium large*.—A head of 22 inches circumference or over may be considered large in any school child; allowance must be made for age. Hydrocephalus was not here included; doubtless many cases were rachitic—364 boys, 59 girls.

*Cranium small*.—The point of size is recorded as apart from the height of the child—476 boys, 1254 girls.

*Cranium bossed*.—There may be bosses, protuberances, or outgrowths at the site of the ossific centres of the frontal and parietal bones—818 boys, 174 girls.

*Forehead defective*.—The forehead may be narrow, shallow in vertical measurement, or small in all dimensions; it may bulge forward and overhang. All defects of forehead except “bosses” and “frontal ridge” are here included.

*Interfrontal ridge*.—The vertical suture between the two halves of the frontal bone may be the site of a bony ridge, present in all degrees; if the forehead is also narrow, it forms the scapho-cephalic type (6), which is the worst form of cranial development—210 boys, 46 girls.

Four principal types of head may be mentioned: dolicho-cephalic or long head; oxy-cephalic or the elevated head; brachy-cephalic or broad-headed, and micro-cephalic, with a circumference below 17 inches after the second year. Dr. Arthur Macdonald says: “Bright girls have a larger percentage of long-headedness than dull girls; the reverse is the case with boys.” See *Experimental Study of Children*, p. 1005 (3).

The significance of cranial defects is shown by the conditions correlated, as follows:—

	With Abnormal Nerve Signs.				Delicate or Thin.				Mentally Dull.			
	No. of Cases.		Percentage of Cases.		No. of Cases.		Percentage of Cases.		No. of Cases.		Percentage of Cases.	
	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.	Boys.	Girls.
All cranial defects	850	531	55·6	50·6	392	480	25·7	45·8	634	477	41·4	45·5
Cranium small .	177	372	54·1	50·4	151	399	46·1	54·1	165	353	50·4	47·8
„ symmetrical	40	7	47·6	43·7	18	3	21·4	18·7	35	6	41·6	37·5

A *small head*, that is of circumference of 19 or less than 20 inches after



seven years of age, is a defect—contrary to the rule—more frequent among girls than boys. There may be average mental ability, but the child usually remains thin and delicate, with liability to exhaustion (9).

The *palate*, or space between the teeth of the upper jaw, may be defective in form and proportions, as seen in the vertical and horizontal plane. The alveolar processes, in place of being parallel at the site of the molars and forming a semicircle above the canine and incisor teeth towards the front, may form two straight lines, meeting in an acute angle in front, the palate between them being contracted—that is, it is V-shaped in the horizontal plane. Again, the palate may be highly arched, or vaulted with a Gothic roof, and at the same time contracted laterally. It is not uncommon in observing physiognomy to see a lateral contraction of the superior maxillary bones, leading to sinking in of the sides of the face below the cheeks, while inspection of the mouth shows smallness of the palatine plates and a narrow palate. Subnormal conditions of the palate are almost as much correlated with abnormality of the individual as defects of the cranium in other parts; they are often associated with adenoid growths and nasal obstruction.

The features should be observed separately, and bilateral parts compared.

The *nose* is constructed partly of bone, the anterior portion being formed in cartilage; these parts should be observed separately. The bony bridge of the nose may be wide and thick; this is often accompanied by obstruction of the respiratory passage. The nostrils may be small, further impeding the air-passages.

The *ears*, like other parts, grow, and should be of a size recognisable as proportional to the head. The normal character seems to depend upon the presence of all parts—the helix, anti-helix, pinna, and lobule. The most frequent abnormality is a large outstanding ear, with defect of the helix and absence of the anti-helix, while the skin is red with varicose venules and adherent to the cartilage.

The *palpebral fissures* may be small though well shapen, leaving but a small space between the lids; the transverse axis from the canthus to the external angle, in place of being horizontal, may slope outwards and downwards or upwards.

The *mouth*, as a feature at rest, should be of sufficient size, and the lips thin, without enlargement at the margins of the mucous membrane. A small mouth is the most usual defect, often accompanying a narrow palate and respiratory obstructions.

The *features collectively* may be heavy and flat; parts may be small or large and ill-proportioned. The separate features may not be individually malformed, but disproportionate to one another or to the size of the face; thus, the nose may be small while the face is large, round, flat, the features rising from the plane of the face; a small mouth with small eye-openings gives a blank look. The face, with the upper and lower jaws, may be small, independent of the size of the calvarium and brain-case. As a criterion of the normal development of a feature all its parts should be present; for instance, in the ear, the helix, anti-helix, pinna, and lobe should be looked at as well as the texture of the skin covering it.

Other defects in development might be described, and the proportions in growth of the body might be referred to, but this belongs rather to the subject of anthropometry than physiognomy.

The methods of studying observations of physiognomy, or recorded facts, are important, and should be considered in their relations to other con-



ditions associated with them (17). It is particularly when dealing with questions as to the constitution or congenital status of the brain of an individual that observation of the physiognomy or development and the expression or neuro-muscular action together give so much aid. The careful *study* of physiognomical signs and conditions of development, rather than new observations, has afforded recent advance in knowledge; the records of the state of general nutrition of the body, together with the neuro-motor signs of brain action, have been closely studied in their inter-relations, together with other facts in physiology. Various conditions of bodily development may be studied among themselves, and also in their relations with other causes of a normal or subnormal state, thus adding to our knowledge and experience in many ways, and throwing a new light on conditions of evolution, reversion, laws of nutrition, causes of infant mortality, and other matters of interest in relation to diagnosis and etiology.

Physiognomical signs, that is points in developmental growth, prove nothing themselves with regard to the brain, except the obvious fact that when the head is small the brain which it contains is small also. Still they suggest probabilities of value in pathology and prognosis. When developmental defects are seen coincident with actual signs of brain deficiency, they indicate the probability of congenital origin.

Summary and generalisation of observations have shown many new facts, among others, the frequent coincidence of several developmental defects in the same individual. It is largely in connection with the diagnosis of causes of delicacy and low nutrition, as well as brain disorder, that the study of physiognomy is most useful in clinical medicine (18). Classification and analysis of the physiognomy in a large number of cases have shown most clearly the greater frequency of developmental irregularities in males, while the low nutrition and delicacy accompanying are greater with females at all ages.

Sometimes we may recognise reversion in the type of development, as the Mongolian type, when the palpebral fissures are narrow and slope downwards and outwards. The frequency of this form of development in some idiots has been shown by Dr. Langdon Down (2) and other observers; it is also not infrequently seen among persons of normal brain-power.

Prenatal development may be arrested, as in cleft-palate and hare-lip, visible remnants of the branchial clefts and other points.

Occasionally supernumerary parts are developed, such as rudimentary ears, extra fingers and toes. I have not found these and the like deformities much associated with defect of the brain. Development may be late for the age of the child; thus, the bony portion of the nose, the bridge and septum, are not much developed in the infant; the proportions of growth change later on, so that the feature becomes prominent when five or six years old. This forward growth of the nose may be delayed, not occurring till seven years or later, without disease; such may be seen in several members of the same family. Teething is often delayed in children of backward development, and the third molars are often imperfectly developed. In all cases of delayed development it is advisable to take the weight and height, together with a few anthropometric measurements, for comparison with the normal of the age (19).

Expression and movement in the face, and by gestures and attitudes of the head, limbs, and body, have most frequently been described as indicating emotions. The practitioner of medicine needs physical signs of action in the central nerve-system, such as may be observed singly or collectively;



he can thus proceed with his investigations, as when using physical signs indicative of a normal or subnormal state of the heart and lungs or other organs, interpreting what is seen, and making inferences from observations. These nerve signs may be normal or subnormal; each must be recognised, that health and disorder occurring in varying degree may be appreciated. It is the neuro-motor signs of brain action that we study in expression.

In describing any expression of brain status, as seen in movement, the part moving must be indicated, whether in the face or hand, etc., as well as the direction of the movement, and if possible its degree, also its relations to any antecedent obviously connected with it, such as a sensory stimulus. We should not speak of movement as voluntary, purposive, or intelligent, but as following auditory or visual stimulus, repeated as formerly seen, apparently spontaneous, or in other terms indicating what we observe (7).

*The face* in normal expression moves symmetrically. In the forehead the frontal and corrugator muscles produce action, seen in the tone and wrinkling of the skin; thus horizontal and vertical creases respectively are produced. Frowning (horizontal creases) may be fine or of course degree; it may occur spontaneously, mostly corresponding to an unoccupied and inert mental state, and is more common in boys than girls. This action may present a uniformly repeated movement, athetoid in character, recurring spontaneously or following every stimulus. Knitting of the eyebrows (corrugation) may be a fine movement indicative of mental stress; it may also occur with frowning as a spontaneous or athetoid action. Muscular tone about the lower lid (*orbicularis oculi*) is present in a lively face; it is increased in smiling, but lessened in fatigue, bodily or mentally, so that the lids look puffy or swollen, as is common accompanying migraine (16). Important items of expression are seen about the angles of the mouth, which move upwards and outwards in smiling, with increase or duplication of the nasolabial grooves, or with convulsive spreading movement in laughter, or coarse repetitive action in grinning. Expression in the upper lip may be asymmetrical, uncovering the canine tooth, as in sneering, also during speech in a general paralytic, accompanied by tremor, and in the spasm of stammering which spreads in the face. The angles of the mouth are depressed in physical pain, the line of the lips being arched downwards at the commencement of crying; this expression is antithetical to that of joyfulness, and there may be a struggle between the expression of the two emotions (12).

*Movements of the eyeballs*, like other action, should be under control of the brain centres, and readily co-ordinated through sight or hearing as well as acquired habit (15-19). Expression in the size and reaction of the pupil is too large a subject to deal with here. Eye movements are often spontaneous when similar action is seen in the face and the hand. Well-controlled movements are seen when the eyes are easily directed in following a moving object without turning the head, and similarly in looking at and counting objects while the head is kept quiet. Spontaneous eye movements characterise neurotic conditions and chorea; when chiefly upwards in direction they may indicate mental excitement; lateral oscillations are often seen in mental emotion and in the insane, as apart from organic nystagmus.

*The hand*, like the face, expresses brain action in its balance and movements. The hand when held out promptly, without extra movements, should balance straight, all parts in the same plane, without folding of the metacarpus or drooping of the thumb or fingers, while the arm is straight at the elbow and held on a level with the shoulder. This attitude expresses



well-trained brain balance, while co-ordinated movements of the digits in response to direction or imitation, accurate in the time and degree of each act, whether in flexion or in lateral movements, as performed by the small intrinsic muscles of the hand, show good brain response. In nervous subjects the metacarpus is arched, and its bones drawn together, making it look narrow and concave on the palmar surface, while the knuckle-joints are extended backwards and the thumb bent outwards. This is often accompanied by finger twitches, either flexor-extensor or lateral, when the digits are separated. These points are usually seen in a child convalescent from chorea.

In fatigue and listlessness the hand is held out more slowly and at a lower level than the shoulder, the palm contracted as before described, with the wrist and fingers drooped. This, like the nervous balance, is usually more marked on the left side.

The movements of expression may be described and classified for further study as occurring in large or in small parts of the body. Rotation of the head to one side, with raising of the scapula; jerking movements of both shoulders while they are brought somewhat forward; movement at shoulder-joint and elbow; shifting the lower limbs at the hips, or binding the knees, are examples of large muscles in action; these frequently occur in functional and so-called hysterical cases. These differ in the signification of their expression from the finer movements of the digits, the eyeballs, and the mobile features of the face, and the tongue, which more directly represent action in the higher centres, and are often expressive of mental states; they are readily interfered with by local changes in the brain, such as general paralysis at its commencement, chorea of slight degree, and neurotic disturbances. Further examples will appear in speaking of classes of movements.

In describing expression, besides indicating the parts that move, whether small or large, the character and relations of series of movements are of importance. Movements may be classified or grouped without reference to either the parts moving or the cause of the action seen.

*Uniformly repeated movements* of the same parts recurring in the same order, when spontaneous, have been called automatic; such may be seen in repeated frowning, grinning, grimaces, shoulder shrugging, tricks and habits, such as protrusion of the tongue, tapping with a finger on the table, as well as in athetosis. Such movements are unintelligent, and frequent among imbeciles.

*An increasing area of movement* may express emotion, and follow a slight stimulus causing much brain action, such as a few words or even a look from another person. Action thus spreads over the face in smiling. In laughter it is stronger; commencing about the mouth, the whole body is finally convulsed; the spreading convulsion of passion and epilepsy lead to brain exhaustion; in chorea a slight stimulus produces widely-distributed movement. A spreading movement may be uniformly repeated on various occasions, as the area of spasm in stammering.

*A lessening series of movements* is seen when excitement or spontaneity lessens, as rest or sleepiness supervenes; also when fidgeting is partially inhibited during mental attention, as well as during convalescence from chorea, and when an epileptic or hysterical fit or storm of passion subsides.

*A co-ordinated series of movements* expresses a relation to the environment in its present or past control. One act succeeds another, not an increasing area of movement; each act accomplishes something in well



co-ordinated action, and the series follows a certain order. Co-ordination is usually less fatiguing than the spreading expression of emotion.

Expression may be described by indicating individual neuro-motor signs and series of movements; besides this, it is useful to study expression of the general characters of the state of the brain, that is, the modes of sensory response, which may be conveniently grouped under headings.

*Spontaneity* is the normal character of the young brain. This kind of movement may be seen in infants, and is abundant in childhood; usually there is variety rather than repetition in such action. The area of movement may spread or diminish under observation; then we infer a corresponding increase or decrease of spontaneity in the nerve-centres. In early years spontaneity is seen in uncontrolled movements of all parts of the body, especially the digits and the eye-balls, while disconnected thoughts similarly arise, but later they become controllable by training. Spontaneous movement lessens towards adolescence, remaining latest in facial expression and in speech, while spontaneous thoughts continue more or less to the end of life. Uncontrolled movement may occur in fatigue or low health, as fidgetiness, restlessness and chorea, or as mental action in delirium.

*Impressionability* is shown by any of the modes of expression in which action is controlled by the environment through the senses, whether the impression be permanent or not.

*Inhibition* is expressed when, in an infant three or four months old, the spontaneous movements are momentarily arrested after an impression by sight or sound, at first for a few seconds only. At this stage of brain evolution we cannot infer what occurs in the brain during the pause, but at, say, five months old, co-ordinated action follows the arrest of spontaneity; the object seen is grasped, or the head and eyes turn towards the sound; the brain is prepared for this co-ordinated action during the pause. An older child, when asked a question, tends to repeat it; the fidgeting is probably arrested in part, then comes a pause for thought followed by expression in words. We infer that some readjustment of the brain-centres (psychosis) (12) occurs during the period of inhibition of movement corresponding to thought.

*Control through the senses* expresses the reaction of the nerve-centres to external stimulation; this should be tested separately for hearing, sight, and other sources of impression, and the effects noted in controlling spontaneity and in co-ordinated action.

*Control through muscle sense* is an important means of impressing the brain; muscular contraction sends a stimulus up to the brain, thus producing effects often mental in character. Muscle sense is exerted in two ways—(1) Contraction of muscles leads to mental action and expression when objects are counted by eye-movements, or their size is felt by the fingers as apart from sight, when length and measurements are felt; also in touching parts of the face or indicating the position of the limbs. (2) Tension of muscle produces expression after feeling and comparing weights held in the open hand, when the degree of strain on the muscles is expressed in words, or when the degree of pressure made by the fingers is indicated.

*Compound Cerebration*.—Expression by movement, or by words following a single sensory stimulus, may be complex, and so far different from the primary impression received as to indicate clearly that it is not an act of simple imitation or repetition, but is due to interaction among the brain-centres. In imitation the centres corresponding to those active in the brain of the person imitated come into activity one after another, guided by each movement seen as it occurs. In the complex action referred to we



must infer that the primary stimulus is followed by nerve currents passing from certain nerve-cells to others in turn, to be finally succeeded by expression in action well adapted by the sensory impression. The relation of the primary stimulus to the final action can only be interpreted as an interaction among the brain-centres occurring during the pause for response.

*Retentiveness* is expressed in repeating a movement exactly as it was performed on a previous occasion; when this follows a direction given, the action indicates some memory and intelligence. Any act of memory is due to the revived activity of impressions previously received and retained in the nerve-centres producing expression by words or actions.

*Co-ordinated action* is expressed when a series of acts is obviously controlled by the environment, as by a direction given, or when it so far resembles similar action performed previously as shows it to be due to past impressions and acquired experience. Thus a number of acts follow one another, due either to immediate sensory control or to the interaction of the nerve-centres among one another, suggesting their connection by nerve pathways previously formed.

*Spreading area of brain action* is expressed by an increasing area of movement seen, and probably indicates a greater discharge of force from nerve-cells than co-ordinated action does. We have no unit of measure for nerve force expended in action; it seems probable that the nerve energy spent is partly that proceeding to the muscles, and in part that producing new nerve pathways, as to the physiology of which we are ignorant.

*Response* may immediately follow the stimulus eliciting it, or there may be a prolonged pause of inhibitory action; there is usually no delay in simple motor response, but a pause for thought (attention) is common, and it may be inferred that during the interval of time there is a process of interaction among the brain-centres (compound cerebration), sometimes accompanied by spreading brain action (mental confusion). Motor response is seen in reflex action without delay; this is similarly repeated on application of the same stimulus; a higher mode is evidenced when co-ordinated action follows a simple direction or command without further guidance.

The study of expression, movements, and individual nerve signs, enables us to give descriptions of what may be seen indicative of various brain conditions, while physiology and the principles of cerebral localisation enable us to interpret these observations and draw inferences from the facts. In such descriptions we say what we look at and what we see in the face and the hand, etc., then classify the neuro-motor signs seen in succession, and proceed to record the general character of the brain status.

Physiognomy and expression, when studied in association with the brain power and health of the individual, show relations among these conditions of great clinical interest (8-10). The facts here presented suggest principles by which we may be guided in making inferences as to modes of brain action in various mental states, as well as in conditions of brain disturbance. We need to be acquainted with (1) the frequency of occurrence of each developmental and nerve sign, and (2) their correlations in age-groups for each sex; such facts have been given earlier in this article concerning defects of the cranium, and tables affording similar information as to other developmental conditions and nerve signs I have given elsewhere (13). The correlations are sufficiently constant and uniform to be useful as guides to prognosis; they vary with sex often to a marked degree, and also in the age-groups among children. The more unusual or infrequent developmental conditions are often the most abnormal, as indicated by their higher correlations.

The intimate relations that have been shown to exist between conditions



of development, health of body, and the modes of brain action, indicate the usefulness of studying physiognomy and expression as aids to diagnosis, etiology, prognosis, and pathology. Children of the neurotic type are usually marked by good physiognomy, with a tendency to spreading movement, much varied in the special nerve signs occurring; the action may be spontaneous, as in chorea, or easily excited, as in mental irritability. On the other hand, low types in development and defects of individual parts, accompanied by repetitive movements—frowning, grimaces, protrusion of the tongue, and repetition of the question asked—are frequently indicative of a brain slow in mental action and imperfect in all response. The clinical description of nerve signs enables inferences to be made as to the neural processes occurring in the brain. Repetitive actions often characterise conditions of hysteria, and are seen in fixed abnormal habits, spasmodic torticollis, and athetosis; emotion, hysteria, chorea, epilepsy, are characterised by spreading brain action, hence improvement results from training and education adapted to bring the brain under control and guidance. In similar manner we may study the neural states corresponding to brain fatigue, sleep, mental confusion, forgetfulness. The observer who habitually compares the attitudes and movements seen in the two hands will soon be convinced that the early indications of inertness and fatigue are more commonly apparent in the left upper extremity, which is so often held at a lower level than the right, while this hand more commonly shows the expression of feebleness or nervousness. Observation of nerve signs thus shows the greater liability of the right cerebral hemisphere to weakness and early fatigue (15-19). Infants with scapho-cephalic heads and premature ossification of the fontanelles are typical of low development; these are more usually met with among males; they are very liable to be cut off by any pulmonary or gastrointestinal disturbance, or by convulsions. Infant mortality is very heavy, especially among males; the registrar-general has shown that the proportion of infant deaths registered under "Developmental diseases" has increased for many years. Among children at school age the report (4) shows that "children with developmental defects" amounted to 13·4 per cent of the boys and 9·6 per cent of the girls, and that a large proportion of these showed subnormal nerve signs and mental dulness, while 16·2 per cent of the boys and 26·3 per cent of the girls were pale, thin, or delicate, the proportions being highest at seven years and under. Many infants of low development and with small heads die of marasmus, others with bronchopneumonia and whooping-cough; these cases add greatly to infant mortality among males, while more girls survive. Among the developmental cases that grow up the males remain most numerous, but the females suffer the most; the proportion of girls with ill-health and mental dulness increases with advancing years, and seems to lead to more permanent lowered capacity.

To illustrate the usefulness of including descriptions of physiognomy and expression in the clinical record of a case, I conclude with the following example quoted from my recent work, *The Nervous System of the Child*.

#### A CHILD OF NERVOUS TYPE.

*Age last birthday*, 14 years.      *Name*,      Girl.

##### A. BODY: DEVELOPMENT, FEATURES, ETC.

*Head*.—Of good volume and well shaped; circumference 21·5 inches. Forehead broad and high.

*Face*.—Features in good proportion. Eye-openings and mouth of sufficient size.

*Ears*.—Well made in rim and pleat of the ear, alike on either side.



*Nose*.—Normal ; breathes with lips closed.

*Palate*.—Sufficiently wide ; good teeth, not crowded.

*Growth*.—Height 60·5 inches (average for age, 60·32 inches). The body well proportioned ; hands and feet rather small.

#### B. NERVE SIGNS : POSTURES, MOVEMENTS, ACTION.

*General balance of body*.—Does not stand straight or keep quite still. Shoulders not at same level ; feet unequally planted.

*Expression*.—Bright and changeful ; a spreading smile often seen, and sometimes twitching of the mouth.

*Orbicularis oculi*.—Want of good tone about the lower eyelids, but this disappears when interested and in smiling.

*Eye-movements*.—Can fix eyes well, but they often wander when not directed.

*Head balance*.—Head not bent down, but often falls a little to one side, or is turned about.

*Hands*.—Held out promptly in response, the left a little lower than the right, while neither is on a level with the shoulder. Each balances in the “nervous posture,” especially the left ; this becomes more marked if the effort is maintained ; there are some twitchings of the fingers. This action is accompanied by some bending of the lower part of the spine, while the shoulders are thrown back.

*Response* is prompt, action is quick, and well imitated from others, but is often accompanied by some extra-movements, besides those under control.

#### INDICATIONS OF MODES OF BRAIN ACTION.

*Spontaneity*.—Fidgets while standing, feet shuffle, fingers twitch. The head is often turned about, the eyes wander, she smiles frequently, and is active in play.

*Impressionability*.—Quick to receive all impressions ; looks at every one who speaks in the class ; is not always completely under control.

*Inhibition*.—While prompt to stand when directed, there remains some fidgeting of the hands with the dress or hair ; she is never quite still.

*Control through senses*.—Good capacity ; but sometimes listens and looks about instead of seeing the blackboard or map demonstration. At times starts or fidgets when spoken to.

*Muscle sense*.—Appreciates and compares weights in the hands well ; knows coins by feeling them. Estimates dimensions better by feeling with the hands than at sight.

*Compound cerebration*.—Physical exercises well performed ; can lead the class without being guided. Proceeds systematically to examine and describe a flower as previously taught. Generally repeats a lesson correctly.

*Retentiveness*.—Memory really good ; but forgets where to find things from not looking to see when putting them away ; can retain facts learnt, but does not always use them aright.

*Co-ordination*.—Imitates hand movements well, but is not quite accurate ; such action is often accompanied by some extra-movements. Speaks well. Good at games.

*Spreading area*.—Extra-movements with the pen are seen before writing ; while at times the fingers twitch on the pen. The head often turns upwards while thinking, or is held on one side when speaking, or when the hands are held out. She tends to laughter and talkativeness. Sometimes there is confusion in replies ; facts of history are remembered, but given in the wrong place.

*Response*.—Quick both in action and in words ; generally without a pause for thinking.

C. PHYSICAL HEALTH AND NUTRITION.—Not pale, but a little thin for her stature ; weight 98 lbs. Average weight for age is 100·32 lbs., but this child is a little above the average height ; further, she has probably not yet completed her growth, which last year increased by 1·75 inches, as against an average of 1·57 for her age, while weight increased only 8 lbs. as compared with the average, which is 9·14 lbs.

*School Report*.—In disposition affectionate ; sometimes loses self-control, and is emotional or passionate.

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## Pica, or Dirt-Eating.

PICA (*pica*, a magpie), dirt-eating or “perversion of the appetite,” are names applied to the habit of eating or putting into the mouth various indigestible or disgusting substances. The habit is most frequently noted in hysterical or pregnant women, the insane, imbeciles, and abnormal children.

In some countries dirt-eating is not confined to the above-mentioned classes, as in the Southern States of North America, Africa, and the West Indies, both children and adults are addicted to eating the clay found on the banks of rivers. The same habit is noted in some of the lower animals, as dogs when suffering from worms, and pregnant bitches will eat any filth or garbage.

The capricious appetite of pregnant women is well known, and women who are perfectly normal in their ways at other times will, when pregnant, crunch cinders or dirt between their teeth.

The dirt-eating tendencies of the insane are commonly enough experienced in asylums, and in not a few instances death has resulted from swallowing knives, hair, or stones. Some idiot children will apparently derive pleasure in crawling about the floor and licking the blacking and dirt off the boots of visitors, or in banging their heads against a wall, or in striking them with their hands.

The most interesting forms of pica may at times be noted in children who cannot be classified as idiots or feeble-minded. In a minor degree an abnormal appetite may be seen in children who are suffering from chronic intestinal catarrh, or who perhaps suffer from worms. The appetite of such will be capricious and uncertain; they will refuse their food at meal-times, but when bathed at night will perhaps take a bite out of a cake of soap with the greatest relish.

In the majority of cases dirt-eating is seen in children with abnormal nervous systems, their eccentricities cropping up in other ways besides in the way of relish for dirt, wall plaster, or the wool off blankets. There may be a tendency to masturbation, moral delinquencies, epileptiform convulsions, or marked perverseness or contrariness of character. The perverted appetite which delights in pushing pieces of slate-pencil up the nostrils, chewing blankets, and crunching cinders, is evidence of a neurosis which ought to put parents and teachers on their guard. While care and careful training will in a proportion of cases result in a more normal state of things, the dirt-eating neurosis marks a tendency to an unbalanced nervous system, which in later years may result in a complete breakdown.



## Pigments of the Body and Excreta.

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OF the pigments which contribute to the coloration of the various organs, tissues, and excreta of the animal body, some perform very important functions, whereas others can only be regarded as mere waste products which are thrown out in the urine and fæces. These latter are for the most part products of the metabolism of the most important of the coloured constituents of the body, namely, hæmoglobin.

The spectroscope has afforded very valuable aid in the recognition and study of many of the animal pigments, and especially of those derived from hæmoglobin, and renders possible their detection even when present in quantities so minute that they could hardly be found by other means. However, its indications require to be controlled by chemical methods, especially when it is sought to obtain the individual pigments in a condition of purity.

Many of the animal pigments owe their importance to the parts which they take in adornment; but it must be remembered that many of the most brilliant hues of birds and insects are not of pigmentary origin, but are optical effects, interference colours, due to the structure of the surfaces which display them. Even excretory products are sometimes utilised for purposes of adornment, as, for example, uric acid, which, as F. G. Hopkins has shown, serves as the white pigment of the wings of certain butterflies, the *Pieridæ*.

Important as surface pigmentation is in connection with sexual selection, it is no less important in affording protection by approximating the colours of animals to those of the surroundings amongst which they live, or by offering a conspicuous warning that a brilliantly tinted insect belongs to a species which is unsuitable for food. In man such functions of the surface pigments are less conspicuously important than amongst the lower animals, but the colouring matters of the hair and skin, not to mention hæmoglobin itself, which exerts a pronounced influence upon the surface tints, have no inconsiderable share in adornment.

The dark pigment of the choroid performs an important function in rendering the eyeball a dark chamber, and hæmoglobin and the other respiratory pigments met with in some of the invertebrata are second to no other constituents of the animal body in the importance of the work which they perform, quite apart from any ornamental function.

Our knowledge of the animal colouring matters is as yet very imperfect, far too incomplete, indeed, to permit of the satisfactory classification of the entire group. Many of them, however, may be placed in one of three fairly defined groups. One of these embraces the various pigments known as lipochromes. In a second are included hæmoglobin and its various coloured derivatives; and in the third are classed the dark brown or black pigments which have received the name of melanins, and which are by many regarded as hæmoglobin derivatives.

THE LIPOCHROMES, or fat pigments, also known as luteins, are very widely distributed, both in the animal and vegetable kingdoms. They are for the most part yellow or red colouring matters, all of which are soluble in alcohol, ether, and chloroform, and are usually met with in combination with fats, from which they may be separated, without undergoing decompo-



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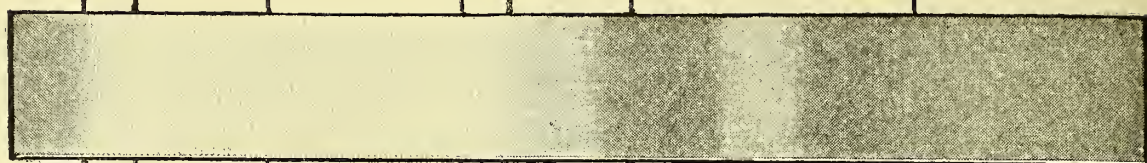
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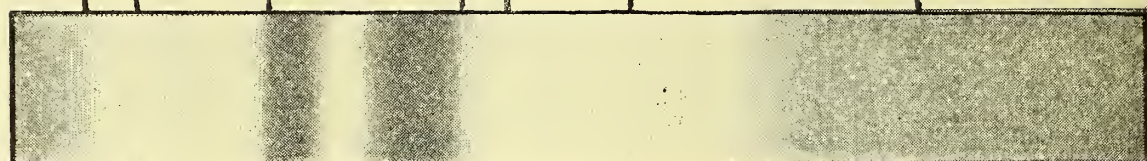
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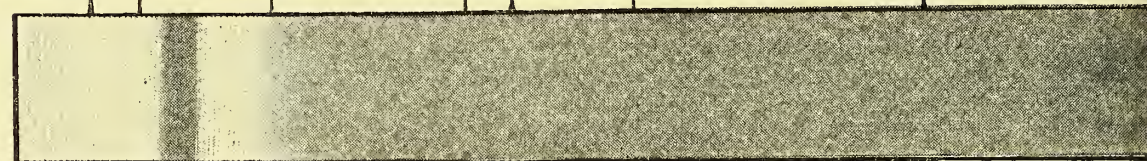
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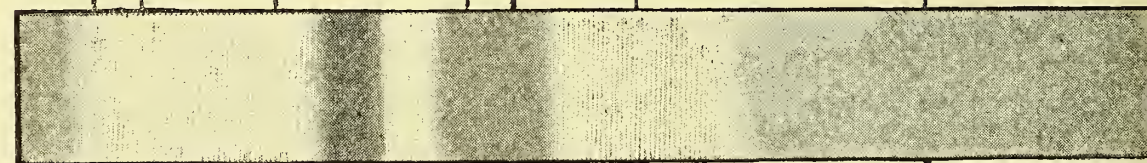
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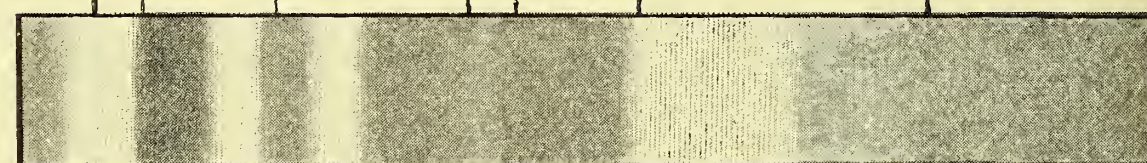
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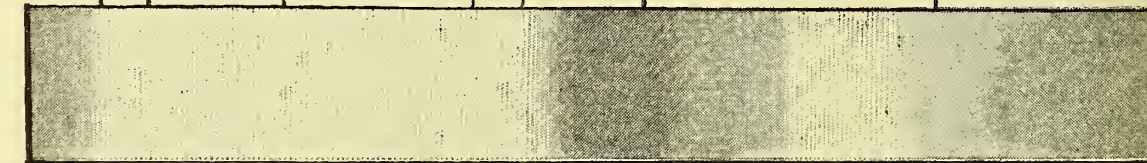
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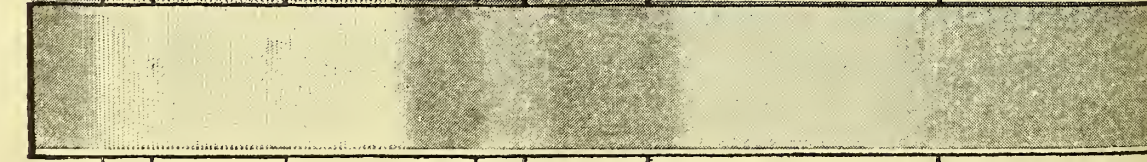
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sition, by the process of saponification. The pigments of this class also agree in being more or less rapidly decolorised by light, with the formation of colourless derivatives which, according to Krukenberg, are allied to cholesterin. In the solid state they yield a blue colour with concentrated sulphuric or nitric acid, and some of them yield a blue colour with iodine, whereas with others this reaction is only very imperfectly obtained. The lipochromes are characterised spectroscopically by showing absorption bands towards the violet end of the spectrum, either a single band near the F line, or, in addition, a second band between F and G.

Carotin, the colouring matter of the carrot, has been more thoroughly studied than any other lipochrome, and this and several other members of the group have been obtained in crystalline form. Husemann assigned to this pigment the formula  $C_{18}H_{24}O$ , but Arnaud has more recently maintained that carotin, as it exists in the vegetables which it colours, contains no oxygen, and assigns to it the formula  $C_{26}H_{38}$ . The absence of nitrogen, at any rate, appears to be a feature common to all the members of the lipochrome group.

In many of the lower animals, such as fishes, reptiles, and birds, the lipochromes play important parts in surface coloration. They impart to the fatty tissues their yellow or orange tints, and it is to members of this group that the colours of the corpora lutea, yolk of egg, and blood-serum are alike due.

A lipochrome which has received considerable attention, and which has a wide distribution in the animal kingdom, is tetronerythrin, which was first isolated by Würm from the red areas around the eyes of certain birds, and has been found by Halliburton to be the red pigment met with in the skin, blood, and hypoderm of the lobster and allied crustacea.

Lutein, the pigment of the corpora lutea, was first described by Thudichum, who thus laid the foundation of the study of this group of substances.

When human fat is extracted with ether the yellow solution which is obtained shows two well-marked absorption bands towards the violet end of the spectrum, due to the lipochrome which it contains, and a similar spectrum is seen when yolk of egg is treated in the same manner. Other interesting members of the group are the chromophanes of the retinal cones of fishes, birds, and reptiles.

The pigment which is mainly responsible for the yellow colour of the blood-serum and of serous effusions is also believed to be a lipochrome. The colouring matter of human serum has received little special study, but from ox serum Krukenberg was able to extract, by means of amylic alcohol, a yellow pigment which showed two absorption bands in the position of lipochrome bands, and Halliburton has obtained similar results with the blood of birds, turtles, and mammalia.

Other pigments may, of course, contribute to the coloration of the serum, such as hæmoglobin, bilirubin, which Hammarsten has shown to be frequently present in the serum of horses, as well as urobilin and other excretory pigments on their way to be excreted by the kidneys.

**HÆMOGLOBIN AND ITS DERIVATIVES.**—In the present article hæmoglobin claims consideration only as a pigment of the body, and as the parent substance of a large family of coloured derivatives. For descriptions of the chemical and spectroscopic properties of these substances, as well as of the far more important respiratory function of hæmoglobin, the reader is referred to works on physiology.

It is a familiar fact that the respective tints of arterial and venous blood



are dependent upon the change of oxyhæmoglobin to reduced hæmoglobin, and *vice versa*. Changes of the colour of the blood are also brought about by the combination of hæmoglobin with gases other than oxygen, as, for example, the cherry tint observed in cases of poisoning by carbon monoxide.

In the surface coloration of the human body hæmoglobin has an important share, and its influence is obvious not only in the visible mucous membranes, but also in the skin itself. An estimate of the part which it plays is readily formed by the observation of patients suffering from extreme anæmia, as contrasted with those who show rosy tints of health; and also from the blue tint of cyanosis, which sometimes attains to so extreme a degree in sufferers from congenital heart-disease. Moreover, when the skin is examined by reflected light the absorption bands of hæmoglobin are clearly visible with the spectroscope.

Methæmoglobin, a compound which has the same percentage composition as oxyhæmoglobin, but in which the oxygen is more firmly held, is often met with in the urine, to which it gives a dark brown or smoky tint. It is not only in hæmoglobinuria that it is present, but also in the ordinary smoky urine of hæmaturia. Hoppe-Seyler stated, indeed, that blood pigment in the urine is always passed in this form, and it is an undoubted fact that blood pigment which has the form of methæmoglobin in the fresh urine may become converted into oxyhæmoglobin on standing, with a corresponding change of tint from brown to pink.

Hæmoglobin also has an important share in the coloration of the solid tissues, partly owing to its presence in the blood contained in their vessels. MacMunn has described a group of pigments allied to hæmoglobin which he calls histohæmatins, the most important of which is myohæmatin, the colouring matter of muscle. He made an elaborate spectroscopic study of this pigment, which he found to be present in the muscles of insects, as well as in those of other invertebrata which have no hæmoglobin in their blood. He also described a modified myohæmatin obtained by artificial gastric digestion of muscle.

On the other hand, Levy maintained that the myohæmatin of MacMunn was not a distinct pigment, but that the spectra observed were due to admixture of hæmoglobin and hæmochromogen, the formation of the latter being due to post-mortem changes.

The most recent investigator in the field is Mörner, who failed to detect in the fresh muscle of dogs and oxen any pigment yielding the spectra described by MacMunn. He observed, however, slight differences between the spectra of the muscle pigment and the blood-colouring matter of the same animal, the absorption bands of the former being slightly shifted towards the red. Similar differences were observed when the corresponding carbonic oxide and methæmoglobin were prepared. On the other hand, the hæmatin from muscle did not differ from that prepared from the blood, and hence he concludes that the difference lies in the proteid components. He proposes for the muscle pigment the name of myochrome.

*Hæmochromogen*, the coloured constituent of hæmoglobin, which has a very characteristic spectrum, has been detected by MacMunn in the medulla of the suprarenal capsules, but otherwise is not known to figure as a pigment of the body.

*Hæmatin* has been met with in urine in sulphuric-acid poisoning, and is possibly present in that excretion more frequently than has hitherto been supposed. It is also found in the alimentary canal, being formed by the action of the digestive juices upon hæmoglobin.



*Hæmatoporphyrin*, an iron-free derivative of hæmoglobin, was known as a laboratory product before its occurrence as a natural pigment was recognised. It has been found by MacMunn in the integuments of certain invertebrate animals, some of which have no hæmoglobin in their blood, and is one of the colouring matters employed in the decoration of the shells of birds' eggs. In normal human urine it is present in minute traces, and occasionally, especially as a result of sulphonal poisoning, it appears in the urine in much larger amount (see art. "HÆMATOPORPHYRINURIA," vol. iv. p. 274). Traces of this pigment are also met with in fæces and bile, and it is more abundantly present in meconium. In a case of sulphonal poisoning it has also been extracted from the blood.

There is as yet no evidence that hæmatoporphyrin is anything but a waste product of metabolism, and the amount excreted appears to be largely dependent upon the condition of the liver.

Hæmatoporphyrin ( $C_{16}H_{18}N_2O_3$ ) is distinguished by the beautiful pink colour of its solutions, and by the distinctive and somewhat complex spectra shown by its solutions under different conditions. The interest attaching to this pigment is greatly increased by the fact that pigments bearing the closest resemblance to it in their spectroscopic properties have been obtained by Church from turacin, and by Schünck and Marchlewski from chlorophyll; but both turacoporphyrin and phylloporphyrin differ from hæmatoporphyrin in their composition. This raises the question how far a practical identity of absorption spectra affords evidence of the identity of the substances which yield them, and, as we shall presently see, the same question arises with regard to the urobilinoid products artificially prepared from bilirubin and hæmatin. On the other hand, such resemblances may fairly be supposed to point at least to a close chemical relationship between the substances from which such allied products are obtained, and probably to the presence in them of a common radicle.

The bile pigments constitute a second class of iron-free hæmoglobin derivatives, and of these bilirubin and biliverdin are by far the most important. Bilirubin has the formula  $C_{16}H_{18}N_2O_3$ , and will be seen to be isomeric with hæmatoporphyrin. In the amorphous state it appears as a yellowish red powder, insoluble in water, very slightly soluble in alcohol and ether, but readily dissolved by chloroform. It forms readily soluble compounds with alkalies, and alkaline solutions turn green on standing, owing to the oxidation of bilirubin to biliverdin ( $C_{16}H_{18}N_2O_4$ ). Unlike bilirubin, biliverdin is readily soluble in alcohol, and insoluble in chloroform. Neither of these pigments shows any spectroscopic absorption bands, but merely general absorption of the more refrangible rays.

The evidence of the derivation of the bile pigments from hæmoglobin is of various kinds, such as their increased formation when excessive hæmolysis is going on; the formation of crystals of hæmatoidin, which is apparently identical with bilirubin, in the seats of blood extravasations; and the fact that under the influence of reducing agents both bilirubin and hæmatin yield urobilinoid products.

The bile of the herbivora is green from the presence of biliverdin, but some doubt exists as to the nature of the pigment of normal human bile. Post-mortem human bile is usually golden or brown in tint, and in gallstones bilirubin calcium is the chief pigmentary ingredient, whereas fistula bile is nearly always of a bright green colour. When the bile pigment traverses the intestine unchanged it may appear in the fæces, either as bilirubin or as biliverdin. When it becomes diffused through the tissues in jaundice it appears in the skin as bilirubin, but in cases of long-standing



jaundice a greenish tint develops. In the urine of jaundiced persons either or both of these pigments may be present, and especially in the urine of infants bilirubin may be deposited in crystalline form.

The other known bile pigments, such as bilifuscin, bilihumin, and biliprasin, have been chiefly met with as constituents of gall-stones. Bili-cyanin has also been found in gall-stones, and cholhæmatin is a pigment with a characteristic spectrum which has been found by MacMunn in the bile of sheep and oxen.

Urobilin is also found in the bile, and appears to be one of a group of allied pigments which may be prepared by the action of reagents upon the blood and bile pigments. It is characterised by a broad dark absorption band near the F line, and by yielding a green fluorescence with zinc chloride and ammonia.

It is abundantly present, chiefly as a chromogen, in normal fæces, and is probably formed by the action of intestinal bacteria upon bilirubin. It differs, however, in composition from hydrobilirubin, the very similar pigment obtained by the action of sodium amalgam upon bilirubin. It is also met with in urine, in traces in health, and here in the form of chromogen, and in far larger amount in many morbid urines which may show its absorption band with great distinctness. There are good grounds for believing that the urobilin of the urine and the bile are alike derived from the intestinal supply, and it disappears from both urine and bile when complete occlusion of the common duct prevents the entry of bile into the intestine.

Among the derivatives of hæmoglobin a place should probably be assigned to urochrome, the yellow colouring matter of normal urine, which has no distinctive spectrum. From this pigment a urobilinoid substance is obtained by the action of aldehyde upon an alcoholic solution, but this, like the products prepared from hæmatin and bilirubin, exhibits differences from the natural urobilin of the urine and fæces. As to the mode and place of origin of urochrome no further evidence is as yet forthcoming.

THE MELANINS.—Our knowledge of the dark brown and black pigments known as melanins is as yet very imperfect. Numerous analyses of such pigments have been carried out, but the results which have been obtained differ somewhat widely, even with materials derived from the same source. The differences observed are specially marked as regards the presence or absence of iron and the proportion of combined sulphur. They are, doubtless, in part due to the difficulty of isolating substances of this description unaltered, but yet in a condition of purity, and in part, perhaps, to the presence of more than one pigment of the group in the same situation. Some observers regard the melanins as derivatives of hæmoglobin, basing their opinion in part upon the presence of iron; but others, including Nencki, take a different view of their origin, and Krukenberg looks upon them as more nearly related to the lipochromes than to hæmoglobin.

In the human body melanins play important parts in the pigmentation of the hair and eyes, as well as of the skin of the dark races, and even in the white races in the coloration of moles. In cases of generalised melanotic tumours large quantities of a pigment or pigments of this class are formed, and may be excreted in part in the urine.

The dark pigment of hair has been investigated by Sieber, who found it to be free from iron, but to contain sulphur. Abel and Davis also failed to find iron in this pigment as well as in that of the skin of negroes.

The black pigment of the retina known as fuscine has been found by Kühne and Mays to contain iron, whereas Scherer and Sieber found neither



iron nor sulphur in the very similar if not identical colouring matter of the choroid coat of the eye.

The melanins of melanotic tumours have been repeatedly investigated. Heintz found no iron in them; Dressler found no sulphur, but a small quantity of iron was detected in the ash. The pigment isolated by Berdez and Nencki, to which they assigned the name of phymatorhusin, was insoluble in water, alcohol, and ether, but readily soluble in alkalies, from which it could be reprecipitated by acids. No iron was present in it, but it contained sulphur in considerable amounts. From melanotic growths in the horse they isolated a second pigment to which they gave the name of hippomelanin.

Mörner, on the other hand, obtained from human tumours, and from the urine of the patient, two pigments, one soluble and the other insoluble in acetic acid. Both contained iron as well as sulphur. He ascribes the absence of iron from the products isolated by previous observers to the employment of hydrochloric acid in their preparation. The sarcomelanin isolated by Schmiedeberg also contained iron, and similar results have been obtained by Brandl and L. Pfeiffer.

The dark pigment present in the urine in such cases is in part or whole passed as a chromogen, melanogen, and darkening may only take place after exposure to the air. Sometimes, however, the urine is of a very dark colour when passed.

Occasionally, as in a case recorded by Senator, the pigment is present in the blood and ascitic fluid as well as in the urine.

OTHER PIGMENTS.—By no means all the pigments of the human body can be placed in one or other of the above groups.

For example, the chemical relationships of the visual purple, the unstable pigment of the retinal rods are unknown. Our knowledge of this substance is largely due to the researches of Kühne.

It is so readily decolorised by light that its separation has to be carried out by the light of the sodium flame. The best solvent is a dilute solution of bile salts, and it can be reprecipitated by removing the bile salts from the liquid by dialysis. It is not only decolorised by light, but also by heating its solutions, by alkalies other than ammonia, by acids, and even by alcohol, ether, and chloroform. The reddish purple solution of the pigment shows no definite absorption bands.

Another pigment which cannot as yet be assigned to any definite class is uroerythrin, the urinary colouring matter which is responsible for the reddy orange tint of febrile urines and that of sufferers from hepatic diseases, and which colours uratic sediments pink. It is an extremely unstable substance, its solutions are rapidly decolorised by light, and it is destroyed by alkalies with the formation of a green product. Its spectrum consists of two feeble bands in the green and blue united by a dark shading.

The indigo pigments are derived indirectly from indol, which is formed in the intestine by the bacterial decomposition of proteids. From the intestine it is absorbed and excreted by the kidneys in the form of indoxyl-sulphuric and indoxyl-glycuronic acids. Indigo blue is sometimes spontaneously deposited from the urine, and in a very few instances renal calculi composed of indigo have been met with. Indoxyl-sulphuric acid is often present in the urine in considerable amount, as, for example, in cases of intestinal obstruction, and under such circumstances the urine, when boiled with hydrochloric acid and a trace of bleaching powder, yields an abundance of indigo blue and red.



Urorosein is a pink pigment which is also obtained by the action of hydrochloric acid upon the urine, from which its colourless chromogen has been isolated in crystalline form. It also is very probably of intestinal origin.

The brown oxidation products of the di-oxybenzenes are also occasionally met with in urine, as in cases of carboluria, and in the rare condition known as alkaptonuria.

Lastly, bacterial pigments must be mentioned, such as pyocyanin, which is the product of the *bacillus pyocyaneus*, and which occasionally imparts to pus a green colour.

In the present article it has only been possible to give a mere outline of the subject of the pigmentation of the human tissues and excreta, and the limits of space have precluded more than incidental reference to the colouring matters of the lower animals which present many problems of great interest. Of some of these pigments copper is an essential constituent, as, for example, hæmocyanin, the respiratory pigment of the blood of some crustacea and molluscs. Turacin, the red pigment of the wing feathers of the touracos, is another copper-containing pigment of much interest. Its properties and composition have been investigated by Professor Church, and, as has been already mentioned, by the action of sulphuric acid upon it a product called turacoporphyrin is obtained, which, in its spectroscopic features, bears the closest resemblance to hæmatoporphyrin.

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**Piles.** See RECTUM.

## Pinta.

**DEFINITION.**—A tropical disease of the skin, characterised by the development of variously coloured patches which later become scaly.

The chief countries in which the disease is frequently met with are:—Brazil, Mexico (especially the provinces of Michoacan and Guerrero), Central America, Venezuela, Colombia, and Peru. It is particularly common in the neighbourhood of creeks and marshes, and is never found at a greater altitude than 2000 metres.

It affects all races, but more especially the half-breeds and those in poor circumstances, while the pure negro seems to enjoy a partial immunity.

**Symptoms.**—The disease begins as one or more small patches usually



situated on the face or extremities, seldom on covered parts. The colour is at first usually slightly red from hyperæmia in white subjects, but in blacks the patch may be fawn-coloured from the first.

The patches rapidly become scaly, and then develop gradually their characteristic colour. This may be a blue or a black in the more superficial varieties, or a red or white in the deeper form. New patches continue to form, while the old ones spread centrifugally, until eventually the whole body with the exception of the palms and soles may be affected. Pruritus is almost invariably severe, and may be noticed even before the appearance of the skin changes.

As the patches become older the skin undergoes more or less degenerative changes, hair turns white and falls out, and the skin shows hard papules, rugosity, and general sclerosis. Probably most of these later changes are due to scratching, since most writers agree that in the chronic widespread cases the itching becomes intense. In the deep variety there is occasionally a discharge superadded, and in the flexures of joints troublesome ulceration may occur. In cases where discharge or ulceration occurs the patient acquires a disagreeable odour, which has been likened to the smell of dirty linen or cat's urine. There are no constitutional symptoms other than those induced by the continual itching, and the disease may, if untreated, persist during the whole lifetime of the patient without serious consequences.

*Etiology and Pathology.*—There still exists some difference of opinion as to the nature of the disease, Lier, of Mexico, maintaining that it is not contagious, after numerous failures in the attempt to inoculate it, and therefore considering that it is rather to be regarded as an abnormality of pigmentation due to attempted adaptation of the skin to the tropical climate. Gastambide, on the other hand, described several varieties of fungus belonging to the order of moulds (*penicillium*), which he found present in the horny layer in the blue and black varieties, and in the deeper layers of the epidermis and corium in the red and white varieties. The fungi varied in the different colours, and Gastambide points out that though the same patient may exhibit several colours at once in different patches, yet a patch of one colour never develops into one of another. The majority of observers seem to agree more or less with Gastambide's views, regarding it as contagious and due to a mould. Inoculation may occur directly from another patient, or possibly by the bites and stings of insects.

Some confusion still seems to exist as to the relation of the disease to those described under the names of Caratés and Cute. Most writers assume that they are identical, but Barbe insists that they are all three separate affections, and says that they may be distinguished by the following characteristics:—Caratés is a slowly progressive disease, taking three years to reach its period stage of full development; pinta is much more rapid, may develop in forty-eight hours, and is extremely contagious; cute is also a disease of rapid development, and differs from both the others in that it is accompanied by fever.

No difficulty should be found in making the diagnosis in patients who come from the affected area. From leprosy the distinction could easily be made by the absence of the characteristic anæsthesia, from Addison's disease by the absence of marked asthenia and abdominal crises, from *tinea versicolor*, *erythrasma*, and *trichophytosis* by the examination of the scales.

The treatment is that of other epiphytic diseases of the skin, *e.g.* unguentum chrysarobini, unguentum hydrargyri nitratis, linimentum iodi.



Long-established cases require great persistence in the treatment, as the disease is extremely liable to occur.

LITERATURE.—1. GASTAMBIDE. *Presse méd. Belge*, 1881, vol. xxxiii. pp. 259, 276, 308.—2. LIER. *Jour. des mals. cut. et syph.* 1897, vol. ix. p. 321.—3. BARBE. *La pratique dermatologique*, Paris, 1900, p. 522.

### **Pituitary Body** (Hypophysis cerebri).

*Structure*.—This body consists of two lobes of different developmental origin: (1) An anterior larger lobe, glandular in type, derived from the embryonic oral cavity; (2) a posterior smaller lobe, belonging genetically to the brain, a continuation of the infundibulum.

The anterior epithelial lobe (hypophysis proper) is light reddish-brown, darker in colour than the posterior one, and in its origin from the oral epiblast is homologous with the buccal glands. It is of great interest histologically, as it has a structure in some respects resembling that of the thyroid. It is made up of numerous tubes which may be only slightly convoluted, and which are arranged in a network. These tubes are held together by connective tissue strands in which there run numerous blood-vessels of wide lumen and with delicate walls. There are also lymphatics between the tubules, which gradually unite to form a network in the capsule. The cells which line the tubes are of two kinds, the one set being larger and more granular than the other. They may contain degenerated red cells or blood pigment, and, according to Benda, fat in the form of globules. A colloid substance is frequently met with in the tubes in the blood-vessels and also in the connective tissue spaces (lymphatics). Fine nerve fibres, sympathetic in origin, have also been described by Berkeley. There have been numerous cases described where the colloid material has occurred in cysts in this lobe, and enlargement of the pituitary has often been noted in cases of acromegaly.

The posterior lobe arises as a hollow downgrowth from the forerunner of the third ventricle; but in the higher vertebrates it is very small and atrophied, consisting mainly of bundles of connective tissue with branched pigmented cells and smaller clasping cells free from pigment. Its original nervous structure is entirely lost.

*Functions*.—Recent investigations have shown that the pituitary, like the suprarenal and thyroid glands, probably furnishes an internal secretion which is of importance in maintaining the normal metabolism. The three methods of investigation which have been employed in attempting to arrive at a knowledge of the character of internal secretions of other ductless glands—namely, that of the *clinician*, where the effects of disease are noted, that of experimental removal, and lastly that of injection of extracts—have all been made use of. The results arrived at, although very valuable, are still not sufficiently definite to warrant one in going further than stating the probable rôle of the body. It is exceedingly unlikely that the pituitary acts in a similar way to the thyroid, although Rogowitsch has stated that in rabbits it enlarges after removal of the thyroid, thus supplying a vicarious secretion, and others have described hypertrophy, tumour growths, etc., in cases of myxœdema. The relationship between enlargement of the pituitary and the occurrence of acromegaly is a more probable one, but their simultaneous occurrence is by no means constant. The experiments of Schiff on excretion of nitrogen and phosphorus, after feeding with pituitary extracts, do not warrant one in drawing the conclusion that the extracts exert an influence on bone metabolism. After complete removal



THE final portion of the article "PLAGUE," by Professor T. R. Fraser, M.D., LL.D., F.R.S., President of the Indian Plague Commission, unfortunately cannot at present be published, and it has been thought better to publish the complete article as an Appendix to the last volume of the *Encyclopædia Medica*, than to either indefinitely delay the present volume or to publish the article in an incomplete state.

*To face page 376, Vol. IX.*







of the gland, animals only survive for a comparatively short period, dying usually within a fortnight. The body temperature is lowered, the animals gradually become feebler, suffer from muscular tremors, and afterwards spasms of a severe nature, and finally pass into a dyspnoëic condition. More exact information was to be expected from investigating the effects of injection of pituitary extracts, and these expectations have been fulfilled by the work of Schäfer and his collaborateurs. The action is quite different from that possessed by the thyroid, as aqueous extracts of the infundibular or nervous portion have a very marked effect upon the blood pressure, the resultant rise being due not only to an increased heart's action, but especially to a direct local action upon the systemic arterioles. This contraction of the arterioles can be demonstrated by circulating a normal saline extract of the pituitary through the blood-vessels of a frog after destruction of its entire nervous system, or by the plethysmographic method applied to the limbs, intestines, or spleen, when in every case there is a distinct diminution in volume. Unlike the suprarenal, however, the aqueous infundibular extract produces, after a brief latent period, a marked expansion of the kidney, accompanied at the outset only by a slight rise in general blood pressure which lasts only a few minutes. This expansion continues for a long period, and concomitant with it there is a marked diuresis, which dies away, however, before the expansion ceases. The substance or substances which produce this effect are not soluble in alcohol; in fact saline extracts of the alcohol soluble constituents of the glands seem to produce in some cases at least the opposite effects—temporary fall in blood pressure, contraction of the kidneys, and diminution in flow of urine. This action is probably, however, not specific, but due rather to depression of cardiac activity, a condition easily brought about in the cat, in which animal the above-mentioned effects are most easily produced. So far, then, as our present knowledge leads us, the specific action of the infundibular part of the pituitary seems to be, in part at least, a renal one. The nature of the active principle or principles requires still to be worked out.

LITERATURE. — Articles by Schäfer, etc., in *Journal of Physiology*. *Text-book of Physiology*, article "Ductless Glands," where the literature on the subject is fully given up to the date of publication.

**Pityriasis.** See SKIN, DISORDERS OF SWEAT AND SEBACEOUS GLANDS.

**Placenta.** See PREGNANCY.

**Plague.** See Appendix to the last volume of *Encyclopædia Medica*.



## Pleura, Diseases of.

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*See also* LUNGS ; MEDIASTINUM

ANATOMY.—The pleuræ are two independent serous sacs which do not communicate with each other. Each sac consists of two layers—a visceral layer and a parietal layer—the former of which completely invests the corresponding lung, including the sulci between the lobes, while the latter lines the ribs and intercostal spaces, invests the upper surface of the diaphragm, takes part in the formation of the mediastinum, and is continued through the upper aperture of the thorax into the neck. The two layers of the pleura move freely upon each other in health. In disease these movements are impeded or rendered painful. The two layers of the pleura are continuous with each other at the root of each lung, and also by means of the *ligamentum latum pulmonis*, a short double layer of serous membrane which occupies the interval between the root of the lung and the diaphragm. The shape of the pleuræ is not the same on the two sides, being determined by that of the corresponding lung. Inferiorly the lungs do not completely fill the thoracic cavity, except in exceptional cases of extreme inspiratory effort.

Only the relations of the pleuræ which are of clinical importance can be here considered. The height to which the pleuræ rise in the neck shows considerable variations. From half an inch to an inch and a half above the clavicle may be taken to represent the usual limits of this variation. In front the pleuræ of the opposite sides are in contact behind the body of the sternum, but the line of reflection is not constant. It is usually behind either the body of the sternum or its left edge. Inferiorly the relations of the pleuræ to the diaphragm are of great clinical importance. According to Quain “the lower border of the costal pleura is reflected on to the diaphragm, opposite a line passing from the lower end of the sternum outwards behind the seventh costal cartilage, nearly as far as its rib ; here it leaves the seventh cartilage, and, continuing to pass obliquely downwards and backwards, crosses the eighth, ninth, tenth, and eleventh ribs, and reaches the twelfth rib near its vertebral end.” Others place the inferior limit of the pleuræ in front, at the level of the sixth intercostal cartilage or the sixth intercostal space. In the mid-axillary line the lower limit of the pleuræ is from two to three inches above the lower margin of the thorax. The inferior limit is usually lower on the left side than on the right, viz. the lower border of the ninth rib on the right side, and the tenth on the left. The inferior limit of the pleuræ posteriorly varies from the upper border of the twelfth rib to the level of the first lumbar vertebra. The relations of the pleuræ to the pericardium are sometimes of clinical importance. “The layers forming the sides of the anterior mediastinum pass backwards from the sternum to the pericardium in close relationship with one another, except below, where a triangular interval is sometimes formed between them. At the front of the pericardium the two layers separate, each passing round its own side of the pericardium to the front of the root of the corresponding lung, where it becomes continuous with the visceral pleura” (Quain). The triangular interval here mentioned corresponds to the inner end of the fifth left intercostal space, and is of importance inasmuch as this is the only situation in which *paracentesis pericardii* can be performed without wounding the pleura. In many cases, however, this space is covered by pleura.



Communications by means of the lymphatic channels exist between the pleuræ and the peritoneum.

**PATHOLOGY.**—The pathology of affections of the pleura is at present in an unsettled state. The tendency of recent investigations has been to regard all pleurisies as toxic, to accentuate the large part in causation which is played by various micro-organisms, to regard most pleurisies not otherwise obviously accounted for as probably tubercular, and to call in question the existence of the so-called “simple,” “idiopathic,” or “inflammatory” pleurisy. These controverted questions will be presently considered in some detail. The bacteriology of the disease has engaged the attention of a large number of observers in this country, on the Continent, and in America, amongst whom may be mentioned Fraenkel, Netter, Weichselbaum, Laveran, Serafini, Levy, Lemoine, Aschoff, Mitchell Prudden, Sidney Martin, Washbourne, Goldscheider, Jakowski, Thue, and Landouzy. The results obtained have been of much interest and considerable practical value, but discrepancies appear in the conclusions of different observers, and it is evident that finality is as yet far from having been attained. The conclusions of pathology, and especially of bacteriology, must be subjected to a rigid criticism and interpreted in the light of the facts of clinical experience before satisfactory views can be attained. In view of the contradictory results of many observers the statement of E. Levy that “die Aetiologie der Pleuritis welche vor wenigen Jahren noch in tiefes Dunkel gehüllt war, heute mit Hilfe der bacteriologischen Forschung ein wohlgekanntes wohl durchgearbeitetes Gebiet geworden sei,” must be pronounced premature. Bacteriology has not yet spoken its decisive word on this subject, but the path of probable fruitful progress has been clearly indicated.

The changes which take place in pleurisy are in many respects those common to inflammations of serous membranes. The membrane becomes injected and hyperæmic, and loses its normal glistening appearance. There is proliferation of the nuclei of the endothelial cells, and soon a fibrinous exudation appears on the surface of the membrane and forms shaggy projections from it. The process may stop at this point, as in the so-called “dry” pleurisy, and adhesions may form between the opposed pleural surfaces; but in a large proportion of cases serous fluid is exuded, and collects between the layers of the pleura. This fluid may consist of limpid serum, or of a more or less turbid and flocculent fluid, or of pus, or it may contain varying quantities of blood. The quantity may vary from a few ounces to many pints. After a variable period absorption usually sets in, and may proceed satisfactorily until convalescence is completed. The two layers of the pleura again come into contact, granulation tissue is formed, and the two layers become in many cases more or less adherent, the pleural cavity being to this extent obliterated. The lung on the affected side tends to collapse, at first owing to the alteration of the normal negative pressure in the pleural cavity, later in consequence of the pressure of the increasing exudation. It may become entirely compressed and airless, or it may float on the surface of the fluid. It tends to shrink towards its root.

We shall now consider the various kinds of effusion in more detail.

**A. Serous Effusion.**—The term “serous” is a misnomer, as the fluid always contains fibrin. It is in reality a dilute liquor sanguinis. The colour is yellow or yellowish green. The specific gravity usually exceeds 1018, and may be as high as 1030, but may also be low. The reaction is alkaline. The fluid may be perfectly limpid, but is often turbid, owing to the presence of large quantities of corpuscular elements. In this latter case the transition to pus is probable, but perfectly limpid serum rarely becomes purulent. Microscopic examination shows the presence of scattered red blood corpuscles, leucocytes, fatty globules, and endothelial cells. Micro-organisms are often present. Sugar is always to be found in small quantity, and sometimes acetone and uric acid. Serous effusions are highly albuminous, proteid material being abundantly present, chiefly in the form of serum albumin, and globulin. Serous effusions coagulate on standing for a few hours. The differentiation of “serous” pleural effusion from passive transudations, due to cardiac or renal disease, may be important. The distinction is not



always easy, and may be very difficult or even impossible. The broad rules are that transudations are usually of lower specific gravity than effusions, they exhibit fewer tissue elements, and coagulate with greater difficulty. (See "Examination of Pathological Fluids," vol. iii. p. 500, *et seq.*)

B. *Purulent Effusion (Empyema)*.—This type of effusion is commoner in the child than in the adult. It is often a sequel of pneumonia, and seems in a considerable proportion of cases the result of invasion by the pneumococcus. It does not often supervene upon a serous effusion unless that effusion has from the outset contained a large proportion of corpuscular elements. It may follow upon repeated tapplings of a recurring serous effusion, but this is much less frequent than has been supposed. The pus only exceptionally becomes fœtid.

C. *Hæmorrhagic Effusion*.—The blood is mixed in varying proportion with serum, less often with pus. Hæmorrhagic pleural effusions are somewhat rare, but their importance is great. They are most often due to tuberculosis, less often to scurvy, hæmophilia, one of the malignant exanthemata, Bright's disease, cirrhosis of the liver, or to cancer. It is important to bear in mind that the effusion in tuberculosis of the pleura is most often serous, only exceptionally purulent or hæmorrhagic. In cancer the effusion is often hæmorrhagic, but may be serous. Apart from tuberculosis, cancer, scurvy, or hæmophilia, a hæmorrhagic effusion points to a state of profound dyscrasia or to a virulent attack of one of the exanthemata. A hæmorrhagic effusion in tuberculosis does not necessarily exclude a fairly hopeful outlook. While hæmorrhagic effusions are distinctly unfavourable, their prognostic indications vary widely and need be interpreted with caution. Cancer cells are sometimes present in a hæmorrhagic effusion. According to von Jaksch, the presence of the glycogen reaction in a marked degree favours the suspicion of cancer.

D. A chylous effusion, due to rupture of the thoracic duct, has been described. It is extremely rare.

BACTERIOLOGY OF PLEURAL EFFUSIONS.—This subject is at present exciting great attention, and may be expected to throw much light upon the pathogeny of pleurisy. The results hitherto obtained are interesting and valuable, but inconclusive. We shall consider the subject under the following heads:—

1. In what proportion of cases of pleural effusion are bacteria present?
2. What kinds of bacteria are found, and what is their significance?
3. What conclusion is to be drawn in cases where the effusion is "sterile"?

As regards the first question we find divergent results. Lemoine found 28 out of 32 cases of serous effusions "sterile." Mitchell Prudden found micro-organisms in only 2 out of 21 cases of pleural effusion. Aschoff found micro-organisms in only 7 out of 200 cases of serous effusion. On the other hand, Thue found micro-organisms present in 12 cases out of 30 in serous effusion, while Jakowski examined 30 cases of serous effusion and discovered micro-organisms in all but 7. It is needless to observe that in cases of empyema micro-organisms are practically always found present.

As regards the second question no constant results have been obtained. Lemoine found the *staphylococcus albus* in 4 cases. He failed to find any micro-organisms in any of his cases which were certainly tubercular. Mitchell Prudden found the *pneumococcus* present in 2 cases of serous pleural effusion following pneumonia. He examined 24 cases of empyema, and found that in "simple" empyema the organism most often present was the *streptococcus pyogenes* (7 times out of 8), while in pneumonic empyema he found the *pneumococcus* in 9 cases out of 11. In one case of tubercular empyema he found the *tubercle bacillus* alone present. Goldscheider found the *streptococcus* in 3 cases of serous pleurisy which did not become purulent, and the *staphylococcus* in 1 case. Aschoff found the *streptococcus* once and the *pneumococcus* once each in 2 cases of serous effusion following pneumonia. In 2 cases of so-called "idiopathic" pleurisy he found the *streptococcus* once and the *pneumococcus* once. In a case of exudation associated with pulmonary gangrene he found the *streptococcus*. Jakowski examined 48 cases of exudation in which micro-organisms were present. In 34 of these only one micro-organism was present; in 10 of these it was the *streptococcus pyogenes*, in 2 the *bacillus tuberculosis*, in 1 the *staphylococcus aureus*; in the remaining 14 cases various micro-organisms were associated. Thue found the *bacillus tuberculosis* present in 1 case out of 12 cases of serous effusion; in the remaining 11 various cocci were found. In 23 cases of empyema he found the *pneumococcus* in 14. Netter examined 110 cases of purulent effusion. He found the *streptococcus pyogenes* in 51, the *pneumococcus* in 32, the *bacillus tuberculosis* in 12, and various bacteria of putrefaction in 15.



Fraenkel, Weichselbaum, Netter, Serafini, and others, assign a very important rôle to the *pneumococcus* in the ætiology of acute pleurisy. Netter, Laveran, and others, regard *streptococcus* pleurisy as much more unfavourable than *pneumococcus* pleurisy. The rarity of the *bacillus tuberculosis*, even in cases undoubtedly tubercular, is noteworthy. Gilbert and Lion failed to find it in any of their 20 cases, though some of them were clearly tubercular. According to von Jaksch, "tubercle bacilli are only present when a discharge of tubercular matter has taken place into the pleural cavity."

As regards the interpretation of those cases of serous effusion which are found to be sterile, there is a growing tendency with bacteriologists to regard them as tubercular. This view rests chiefly on the results of inoculation experiments. Aschoff injected pleuritic fluid from 57 cases of serous pleurisy into the abdominal cavity of guinea-pigs. In 19 of these cases, in which the exudation occurred in connection with pneumonia, rheumatism, and a number of other non-tubercular affections, none of the guinea-pigs became tubercular. In 12 cases of certain tubercular pleurisy the animals became tubercular in 7. In 12 cases, doubtfully tubercular, the animals became tubercular in 9. In 12 cases of so-called "idiopathic" pleurisy tuberculosis followed the injections in 9. The author concludes that the majority of cases of so-called "idiopathic" pleurisy are tubercular. Netter puts the proportion of cases of serous pleurisy due to tuberculosis at 68 per cent, Landouzy at 98 per cent. Germain Sée believes that "so-called simple pleurisy from a chill is only a tuberculous pleurisy, the nature of which has been misunderstood." The whole subject of the relation of pleurisy to tuberculosis—one of the most important problems in practical medicine which are still *sub judice*—can be considered more conveniently after we have dealt with the clinical aspects of the disease. It must suffice here to indicate the general nature of the bacteriological evidence and the consensus of opinion amongst the pathologists that the connection between the two diseases is an intimate one. Jakowski sums up his conclusions on the entire subject of the bacteriology of pleurisy as follows:—(1) That all pleurisies depend upon micro-organisms, though these are not always to be found; (2) That where no organisms are found the case is probably tubercular; (3) That primary genuine pleurisy, not due to tuberculosis, is most often due to the *pneumococcus*; (4) That serous exudations in which pyogenetic cocci are found have a great tendency towards empyema; (5) That exudations during or after pneumonia are most often dependent on Fraenkel's diplococcus. It is evident that these views are still to some degree hypothetical. See "Bacteriology of Pneumonia," p. 409.

*State of the Blood in Pleurisy.*—The blood in uncomplicated cases of pleurisy does not show any very marked changes. Houston, working in Lorrain-Smith's laboratory, found a moderate leucocytosis during the pyrexial stage, the leucocytes numbering from 8000 to 12,000 per cm. This leucocytosis passes off as the fever abates. The same observer found during the afebrile stage the following averages in eight cases of uncomplicated pleurisy:—

Red cells	.	.	.	.	.	.	4,100,000 per cm.
White cells	.	.	.	.	.	.	8,000 "
Hæmoglobin	.	.	.	.	.	.	78 per cent.
Differential count:—							
Polymorpho-nuclear elements	.	.	.	.	.	.	= 67 per cent.
Small lymphocytes	.	.	.	.	.	.	= 28 "
Large lymphocytes	.	.	.	.	.	.	= 2·8 "
Transitional leucocytes	.	.	.	.	.	.	= 1·8 "
Eosinophile leucocytes	.	.	.	.	.	.	= ·4 "

In the febrile stage the proportion of polymorpho-nuclear elements in the differential count is about 70 per cent or more.

Houston is of opinion that the blood-count may be of value in excluding other conditions. In empyema, for example, the leucocytes are usually between 18,000 and 30,000 per cm. In pneumonia a similar condition is found, while in malignant disease of the lung a large leucocytosis has generally been recorded.

In cases of pleurisy the red corpuscles and amount of hæmoglobin usually show only a slight decrease. In cases of chronic pleurisy and empyema the red cells may fall as low as 2,000,000 per cm., and the hæmoglobin may fall to 35 per cent. An empyema may disappear after tapping, and the absence of a leucocytosis has been found useful in determining whether the suppurative process has ceased or not.



## ACUTE PLEURISY

**ÆTIOLOGY.**—The part played in the causation of pleurisy by various micro-organisms—especially the *bacillus tuberculosis*, the *pneumococcus*, and the *streptococcus*—has been already considered. For a further discussion of the influence of tuberculosis see under “Diagnosis,” p. 388.

**Age and Sex.**—Pleurisy is common at all ages. It is not rare in infancy, common in adolescence, attains its maximum frequency in early adult life, and continues somewhat frequent in old age. Of patients dying at advanced ages of various diseases it is the exception to find the pleuræ free from morbid changes. At the Royal Victoria Hospital, Belfast, in the decade 1891-1900, of a total of 9629 admissions 304 were cases of pleurisy, a proportion of 3.15 per cent. There is a preponderance—it is difficult to say to what extent—of cases in the male sex. Hospital statistics on this subject are apt to be fallacious.

**Chill.**—Considerable doubt surrounds the question of the part played by chill in the causation of pleurisy. The writer has inquired into the subject in 74 cases which have been under his observation, chiefly in hospital practice. In 26 a history of chill was obtained, while in the remaining 48 cases there was no such history. In most cases chill took the form either of a wetting or of a rapid cooling of the body when overheated. When it is remembered with what facility a history of chill is volunteered, especially by patients of the hospital class, perhaps the noteworthy fact about these figures is not that a history of chill was available in one-third of the cases, but rather the absence of such a history in two-thirds. Still, the influence of chill cannot be eliminated. Those who hold that all cases of pleurisy are due to the presence of micro-organisms regard chill simply as a predisposing cause, as “lowering the vitality” of the organism, and so rendering it more susceptible to microbic invasion. As regards the influence of chill and its possible relation to the action of bacteria, pleurisy stands on much the same footing as pneumonia.

**Specific Fevers.**—Pleurisy is a not uncommon complication of some of the specific fevers, especially scarlet fever and smallpox. It is somewhat unusual in typhoid fever. Influenza is frequently assigned as a cause—often erroneously.

**Bright's Disease.**—In all forms of chronic Bright's disease we find a proclivity to inflammations of the various serous membranes, and the pleuræ are frequently affected.

**Rheumatism.**—It seems doubtful if there is any real causal connection between rheumatism and pleurisy. Of the 74 cases in the writer's practice rheumatism was noted as being present only in 6. In the case of two diseases which are both sometimes associated with chill this proportion does not seem to exceed the possible limits of accidental concomitance. Valvular disease of the heart was noted in only 4 out of the 74 cases, a fact which tends to corroborate the view that there is no definite relation between pleurisy and rheumatism. It would appear that in the present state of our knowledge there is not sufficient ground for postulating the existence of a “rheumatic pleurisy.”

**CLINICAL HISTORY.**—**Onset.**—In a small number of cases the onset of the disease is acute and “pneumonic” in type. There are grounds for thinking that these cases are due to *pneumococcus* invasion. In a larger group of cases, perhaps about one-half, the onset, though less acute and severe than in the above class, is well marked. The writer found this mode of onset in 18 out of 39 cases. In a third group of cases the onset is



gradual but fairly definite, while in a fourth group the onset is entirely latent, and the disease escapes detection until the patient seeks advice for weakness or shortness of breath, and a pleural effusion of some duration is discovered. The first symptom is usually "stitch" in the side, with embarrassed breathing and some rise of temperature. A series of "chills" is common; a single severe rigor is rare. Vomiting may occur, and in children convulsions are sometimes noted. The temperature rises irregularly, and does not usually exceed  $102^{\circ}$ - $103^{\circ}$  F. In some cases fever is absent. The course and duration of the pyrexia present much variation. The aspect and decubitus of a patient in the early stage of acute pleurisy are often characteristic. He lies on his back, or on the sound side, often curled up in a constrained position in the bed, and he leans forward and presses his hand to the affected side. His face shows that his suffering is severe. In addition to "stitch" in the side there may be a "referred pain" in various parts of the thorax or abdomen. The breathing is shallow and ineffectual, but it is evident that the patient refrains from taking deep breaths not from inability to do so, but simply through fear of exciting pain. There is rarely any of the acute dyspnoea, the *besoin de respirer*, which is so marked in pneumonia. The face is slightly flushed, and in rare cases herpes may be present about the mouth. There are the usual symptoms characteristic of pyrexia.

In a short time, usually within forty-eight hours, the patient's condition undergoes a change. Effusion sets in, the pain in the side is relieved, and the patient feels better, but is conscious of increasing shortness of breath, especially if he makes any exertion. He now finds himself more comfortable lying on the affected side, as this allows the lung on the sound side free play. The temperature remains usually at  $101^{\circ}$ - $103^{\circ}$  F. in the evenings, with morning remission. The patient's condition may now undergo only slight change for some days, the state of the respiratory function depending largely on the amount of the effusion. In favourable cases absorption sets in towards the end of the second or the beginning of the third week, and may proceed rapidly. Pain in the side commonly reappears at this stage. Convalescence may be complete at the end of three or four weeks, but is often delayed. Cough is usually present throughout the disease, but is sometimes absent. It is often hard and unsatisfying, and accompanied by only scanty expectoration, which may be blood-stained at the outset; later it is usually mucoid or muco-purulent. Where œdema of the lung has supervened, on *paracentesis thoracis*, the expectoration is profuse and watery. Foetor of the expectoration usually indicates that a foetid empyema has burst through the lung. Diarrhoea is common. The urine is febrile, and not infrequently contains a little albumin. Attacks of pleurisy present almost every grade of severity, and the symptoms vary accordingly. A tedious convalescence may occur, even in cases which ultimately make a good recovery. If the temperature reach  $104^{\circ}$  F., or the respirations exceed forty per minute, it is probable that pneumonia is present. A hectic temperature and profuse sweating may be present in cases where the effusion consists of limpid serum. Symptoms cannot be relied upon to determine the nature of the effusion (see under "Empyema," p. 394).

In the so-called "latent" or "quiet" pleurisy symptoms are sometimes said to be absent, but evidence of some degree of weakness and shortness of breath can always be elicited.

**PHYSICAL SIGNS.**—The characteristic sign of pleurisy in the first stage, before effusion has set in, is friction, which may, however, be absent. This is a scraping, grating, or creaking sound of variable, but usually considerable,



intensity. Sometimes it has a greasy or sticky quality, and may be difficult to distinguish from crepitus (see under "Diagnosis"). It is usually heard both with inspiration and expiration, but is often loudest towards the end of inspiration. It gives the impression to the ear of being "superficial." It ceases when the breath is held. It is not affected by coughing. It may be intensified by pressure of the stethoscope. Pleural friction is most often heard in the inferior antero-lateral region of the chest or in the axilla. It is also common at the base of the lung posteriorly. If it be audible at the apex the suspicion of tuberculosis is strengthened. It is often confined to a very limited area, but is sometimes heard somewhat widely over the affected side. Friction fremitus is common. While there is at this stage no true flattening of the affected side, the patient expands it as little as possible owing to the pain. The lymph present on the surface of the pleura at this stage does not cause any recognisable dulness on percussion, nor any auscultatory phenomena other than friction.

*Signs of Pleural Effusion.*—Slight effusions may escape detection, but the presence of any considerable degree of effusion gives rise to a characteristic series of physical signs.

(a) *Inspection.*—The affected side is more or less immobile, either in its entire extent in very large effusions, or in a variable proportion of its inferior area. The antero-posterior diameter of the side is increased. The over-action of the healthy side is often marked. The intercostal spaces are usually, but by no means always, flattened. In recent effusions of moderate extent the normal dimpling of the spaces may be preserved for some time. Bulging of the intercostal spaces is excessively rare.

(b) *Palpation.*—In the great majority of cases vocal fremitus is either diminished or lost. In some cases, especially in women and in debilitated patients, the normal vocal fremitus is so weak that the interpretation of this sign becomes difficult, but apart from such cases the decided diminution or loss of vocal fremitus is one of the most trustworthy signs of the presence of fluid in the pleural cavity. It may, however, be fallacious (see under "Diagnosis," p. 386).

(c) *Percussion.*—Increased sense of resistance and dulness co-extensive with the area of the fluid are practically invariable. According to Guttman dulness only becomes appreciable when the layer of fluid has a thickness of at least  $1\frac{1}{2}$  cm. Probably few physicians would now accept Skoda's dictum that "the percussion sound is not altered by mere thickening of the pleura, nor by layers (several lines deep) of fluid or solid exudations upon it." The dulness is not complete until the lung is entirely emptied of air by the pressure of the fluid. In large effusions the dulness takes on a special "wooden" quality, which may be distinguished by the ear from the dulness of consolidation. The dulness usually is first manifest at the base of the lung posteriorly, and in small effusions it may be confined to this region. With an increasing effusion it invades the lateral region and the front, rising to a variable level, and in some cases occupying the entire side. In a considerable proportion of cases the upper level of the fluid is a curved line, the highest point of the curve being often in the axilla (Ellis and Garland). In patients who have been long bed-ridden the highest level of the fluid is usually found posteriorly. The outline of the dull area may be variously conditioned by recent or old adhesions, and may present many variations, which may suggest difficulties in diagnosis. It is quite exceptional for the level of the fluid to be altered by change of posture on the part of the patient. In a doubtful case this test is usually valueless. In the case of a considerable effusion which does not fill the entire pleural



cavity the percussion note below the clavicle on the affected side is often tympanitic. This interesting fact was first observed by Skoda, and hence the sign has been called "Skodaic resonance." Its explanation is doubtful. Skoda's theory was that "when the lung is much reduced in volume by compression, but still contains air, its sound is invariably tympanitic." C. J. B. Williams thought that the tympanitic note was produced in the trachea and the large bronchi, a view which is not, perhaps, very probable.

(d) *Auscultation*.—Enfeeblement or obliteration of the normal vesicular murmur, and weakening, with some alteration of tone of the normal vocal resonance, are the rule, but the auscultatory signs of pleural effusion present great variations, and require to be interpreted with caution. Bronchial breathing is common, especially in children, and may either gradually give way to more or less complete silence, or may continue during the stage of effusion. In the writer's experience the bronchial breathing of pleural effusion has usually had a more or less "distant" quality, which may help to distinguish it from the bronchial breathing of consolidation, but this rule is not invariable. It is probable that where bronchial breathing is present in pleural effusion the large bronchi are still patent.

Vocal resonance presents considerable variations, and should not be much relied upon in diagnosis. In the writer's experience one of the most usual changes is a certain flatness or loss of tone, rather than a mere weakening of the voice on the affected side. Sometimes the vocal resonance is simply weak, sometimes there is bronchophony. The term ægophony has been applied to a certain bleating quality of voice occasionally heard near the angle of the scapula at the upper level of moderate effusions. This sign is rare. It is of small diagnostic importance, and the term "ægophony" might well be dropped.

Bacelli thinks that the whispered voice is well conveyed through serous, but not through purulent effusions (Bacelli's sign). This observation has not been generally confirmed.

(e) *Mensuration*.—In large effusions the semi-circumference of the chest is increased on the affected side, but, as Gee well points out, "by the passage of the elliptical form into the circular, considerable increase in the sectional area of the chest may occur whilst the length of the periphery remains the same. Moreover, the displacement of the mediastinum thrusts the heart into the unaffected side. Add this consideration, too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side, and then it will be plain why, as a matter of fact, the perimeter of the affected side often measures very little more, nay, sometimes even less, than the side which is not diseased." The cyrtometer may be used with advantage.

(f) *Succussion*.—The venerable sign, "Hippocratic succussion," one of the earliest fruits of medical observation, is, of course, only present when the pleura contains air as well as fluid.

(g) *Displacement of Organs*.—Displacement of the heart is the rule, displacement of the liver and spleen the exception, in pleural effusion. The displacement of the heart is more marked in left than in right effusions. The writer has removed 45 oz. of serum from the right pleura in a patient in whom no displacement of the heart could be made out. Such exceptional cases are, however, rare, and great importance must be attached to the position of this organ in any case of doubt or difficulty. Neglect of this rule has been a fruitful source of errors in diagnosis. The usual limits of displacement of the heart are the right mammary line and the left axillary



line, but these limits may be exceeded. In addition to lateral displacement the heart is often raised. Under these circumstances the impulse may be due either to the normal apex or to the right ventricle. Epigastric pulsation may occur.

A systolic murmur is often audible over the heart in pleural effusion, and is probably due to some altered relation between the heart and the great vessels. It disappears with absorption of the effusion.

Upon the unaffected side the movements are exaggerated, and the vesicular murmur is intensified.

*Stage of Absorption.*—After a period varying from two or three days to two or three weeks serous effusions in a large majority of cases begin to undergo absorption. The chief signs of the receding wave are the reappearance of vocal fremitus, if this has been entirely absent, some diminution in the absolutely wooden tone of the percussion note, the gradual return of displaced organs to their normal position, and the reappearance of friction sound and of the vesicular murmur. According to Gee the liquid disappears in something like the following order:—From the vertebral groove near the root of the lung; from the supra-mammary region; from the rest of the vertebral groove and infra-scapular region; from the infra-mammary region; and, lastly, from the lower lateral region. He adds, however, that “we must be prepared to find the residue of liquid in almost any part of the chest.” In the writer’s experience the fluid has often lingered longest at the base of the lung posteriorly, and this is probably the rule in patients who have been much confined to bed.

The rapidity with which absorption is accomplished varies within very wide limits. Sometimes it is fully accomplished in a few days, more often it is somewhat delayed, and not uncommonly it is a tedious and lingering process. Slight dulness on percussion and some impairment of the vesicular murmur may remain for long periods, or for life, in patients who have apparently made a complete recovery. In many cases, however, no trace of the attack remains. The notion that in this latter group of cases permanent adhesions have occurred does not seem warranted by the evidence at present available to us. More or less retraction of the side often remains after an attack of pleurisy. The mythical cases of “people living with one lung” are usually instances of old pleurisies where an extreme degree of retraction of one side of the chest has occurred. In other cases the lung undergoes more or less carnification, and the patient remains permanently short-winded and debilitated, or becomes the subject of tuberculosis.

COMPLICATIONS.—Pleurisy has few characteristic complications. The other serous membranes—most often the pericardium, very rarely the peritoneum—may share in the morbid process. Albuminuria is not uncommon, but is sometimes merely febrile, sometimes it points to a pre-existing nephritis, of which pleurisy is a frequent concomitant. In chronic cases dilatation of the heart and dropsy are not infrequent. Clubbing at the ends of the fingers is common in empyema, but not unknown in chronic cases of serous effusion. Pneumothorax is related rather to empyema than to serous pleurisy (see “PNEUMOTHORAX,” p. 396). Acute œdema of the lungs occasionally, but very rarely, supervenes upon aspiration.

DIFFERENTIAL DIAGNOSIS.—The diagnosis of pleurisy is usually an easy task. Sometimes, however, there may be difficulties and occasionally a positive opinion may be impossible. Difficulties may arise either (*a*) in connection with pleural friction, or (*b*) in connection with the recognition of effusion. We shall consider the subject under these two heads.

Pleural friction is in a large majority of cases unmistakable. Its



rubbing or scraping quality, its superficiality, its dependence on the respiratory act, its independence of coughing, its association with pain, its intensification by pressure of the stethoscope, its usual distribution, make its recognition generally obvious. Its distinction from pericardial friction rarely gives rise to serious difficulty. Pericardial friction is much more rapid than pleural friction, it has usually a to-and-fro quality, it is confined to the præcordial area, and is most frequent at the base of the heart, it does not cease, except in very rare cases, when the breath is held. It must be borne in mind that the reflection of the pleura over the pericardium may be affected, and may give rise to confusing physical signs. A much more serious difficulty arises sometimes in connection with some cases of sticky and greasy exudation on the pleural surface, which may cause a sound readily mistaken for crepitus. The following points will help in obviating this error. Friction is "superficial," crepitus is deep. Friction is not affected by coughing, crepitus often is affected by coughing. Friction is often intensified by pressure of the stethoscope, crepitus never. Friction is often audible throughout the greater part of inspiration and expiration, though there are exceptions to this rule. Crepitus is often, though by no means always, confined to the end of inspiration and the beginning of expiration. Friction is commonly accompanied by pain, crepitus rarely. Lastly, if we examine in the neighbourhood of the doubtful sound we shall often detect unambiguous friction or crepitus, which may settle the diagnosis.

Pleurodynia may be confused with pleurisy. Many cases diagnosed as pleurodynia are really cases of "dry" pleurisy. Error may be best avoided by careful physical examination, and by the use of the thermometer, the presence of pyrexia to be allowed much, its absence little weight.

More formidable are the difficulties which sometimes beset the diagnosis of pleural effusion. Most observers are agreed that in exceptional cases, especially in children, the signs of pleural effusion may be indistinguishable from those of consolidation. As an abstract proposition this is unquestionably true, yet the cases in which the distinction is impossible are not numerous. Too much reliance upon the auscultatory signs is the chief source of error. We should rely chiefly on (1) the state of the vocal fremitus, viz. upon its diminution or disappearance in pleural effusion, its normal or increased intensity in consolidation; (2) the outline and limits of the dull area, sometimes characteristic in pleural effusion (see ante, "Physical Signs," p. 383); and (3) the displacement of organs, especially the heart. If a careful inquiry under these heads still leaves the case in doubt, an exploratory puncture should be made in all cases in which a positive diagnosis is desirable.

In some cases of pneumonia a temporary blocking of the bronchial tubes with secretion may cause signs which simulate those of pleural effusion, viz. loss of vocal fremitus, loss of breath sounds, and diminution or altered quality of vocal resonance. The effect of coughing and expectorating will serve to clear these cases up.

Malignant disease of the lung may give rise to signs which cannot be discriminated with certainty from those of pleural effusion. In a recent case in the writer's practice the lower portion of the right lung presented the signs of wooden dulness on percussion, complete absence of vocal fremitus, and entire loss of breath sounds. The presence of some suspicious superficial nodules and the general history of the case gave rise to the suspicion of malignancy, and at the autopsy the right lung was found to be invaded by a large mass of encephaloid carcinoma. In such cases, which



are of extreme rarity, reliance must be placed, not on physical signs, but upon the history and symptoms. Persistent pain is usually present in malignant disease of the lungs, hæmoptysis is common, but not invariable, the general progress of the case is characteristic of malignancy, and pressure signs tend to develop, but may be late in appearance.

Diaphragmatic pleurisy may give rise to difficulty. Severe pain is the chief feature of these cases at an early stage, more or less pyrexia will probably be present, and after a short time clear physical signs will tend to appear, as the pleurisy is probably never wholly confined to the reflection of the pleura upon the diaphragm.

Encysted or loculated effusions, usually conditioned by the presence of old adhesions, may give rise to puzzling physical signs. Loculation may occur at any part of the pleural cavity, but seems to be most frequent in the lower lateral or posterior region. It may occur between the lobes of the lung. Puncture should be resorted to in these cases, and it is important that the trochar should be inserted at a point where the signs of the presence of fluid are well marked. Otherwise the failure to find fluid may unjustly discredit the diagnosis.

Collapse of the lung, acute or chronic, closely simulates pleural effusion, but the history of these cases is different from that of pleurisy, the side is retracted, the intercostal spaces are not flattened, and the heart is not displaced towards the sound side. Attention to these points may be sufficient to prevent error, but if necessary puncture should be resorted to.

The possibility of subphrenic abscess should be borne in mind in cases which suggest pleurisy, but present abnormal physical signs (see under "Subphrenic Abscess," vol. ii. p. 508, *et seq.*)

The general question of the relation of pleurisy and tuberculosis may be here conveniently considered. The pathological aspect of the subject has been already dealt with. It remains to consider its clinical bearings.

Vincent Bowditch of Boston found that of 91 cases of pleurisy occurring in the practice of his father, Henry J. Bowditch, between the years 1849 and 1879, 32 died of phthisis, or were certainly tuberculous. Fiedler found that of 92 cases of pleurisy with sero-fibrinous exudation, 28 cases were dead of pulmonary tuberculosis within two years, while in many of the remaining cases the presence of tuberculosis was suspected or probable. Barrs of Leeds found that of 62 cases of acute pleurisy occurring at the Leeds Infirmary 22 were dead of phthisis within six years. After an exhaustive treatment of the whole subject, Kingston Fowler concludes that "the bacillus of tubercle is the causative agent in a very large proportion of cases of sero-fibrinous pleurisy, and that this is true when the attack is apparently simple, idiopathic, and attributed to a chill." The same writer further points out: "It must be borne in mind that the course of tuberculosis of the serous membranes is far more favourable than when the disease attacks the lungs and other organs. Of this the clinical history of tuberculous peritonitis affords a striking example. The inference that in many cases of tubercular pleurisy the disease is arrested at the time and never reappears is therefore justified, and is supported by pathological experience." We have seen that Germain Sée regarded all cases of acute pleurisy as tubercular, and that Landouzy from the side of pathology takes practically the same view.

While there is no longer any doubt that the association of pleurisy and tuberculosis is much more intimate than was formerly suspected, there are some considerations which must make us pause before accepting the extreme view on this subject which is now current, especially in France. The onset in pleurisy is sudden in about one-half the total cases. In tuberculosis a sudden onset is the rare exception. Pleurisy following a definite chill, though much less common than was formerly thought, is nevertheless somewhat frequent. Chill does not produce tuberculosis. The incidence of pleurisy and tuberculosis with regard to age and sex shows some points of apparent contrast, but the subject requires further elucidation. Pleurisy most often appears to begin in the lower antero-lateral region of the chest—*i.e.* in that part of the side least protected by muscle—and



shows none of the preference for the apical region which is so marked in pulmonary tuberculosis. The course of the temperature in pleurisy is unlike that of tuberculosis. The general progress of the disease in pleurisy and the early and favourable convalescence of the large majority of cases are unlike tuberculosis. It will be admitted that the clinical course of acute pleurisy bears only a distant resemblance to (*e.g.*) that of tubercular peritonitis. While these points must be borne in mind before we can formulate a definite conclusion on this most important and difficult subject, probably most physicians will agree with Osler when he says : "I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin." It has been seen that the *bacillus tuberculosis* is very rarely present in serous effusions, and the theory that all "sterile" effusions are tubercular is still only an hypothesis. Much will depend upon the results of further inoculation experiments. If it can be conclusively proved that inoculation with serous pleuritic fluid produces tuberculosis in a large and fairly constant proportion of cases the conclusion to be drawn will be obvious. It should not be forgotten that the fact of a patient who has recovered from an acute pleurisy subsequently developing tuberculosis does not prove that the pleurisy was tuberculous. At the most it only raises a presumption that such may have been the case. But it is at least arguable that in some of these cases—perhaps a considerable proportion—a non-tuberculous pleurisy, not thoroughly recovered from, may have weakened the resisting power of the lung, and so rendered it more susceptible to invasion by the bacillus of tubercle. Such a sequence of events is entirely consonant with our present knowledge of the causation of pulmonary tuberculosis.

If pleurisy occur in a patient whose general health is from any cause debilitated, or whose history suggests the probability of antecedent pulmonary mischief, the suspicion of tuberculosis will be much strengthened. In such cases the sputum, if obtainable, should of course be examined with special care.

PROGNOSIS. — The mortality during the acute stage of pleurisy is insignificant. In 39 consecutive cases in the writer's practice death took place in two instances: in one from meningitis, in the other from heart failure consequent on delirium tremens. In neither case did the fatal issue seem to be directly dependent upon disease of the pleura.

Pleurisy derives its great importance almost entirely from three considerations, viz. (1) It may lead to empyema or chronic serous effusion; (2) It may permanently damage the lung and lead to prolonged or permanent ill-health; (3) It may be either a manifestation or a cause of tuberculosis. Our anxiety with regard to patients suffering from pleurisy is chiefly concerned with the possibility of a lingering and unsatisfactory convalescence and of sequential disease. It is important to bear in mind that, on the one hand, tedious and troublesome cases sometimes ultimately make an excellent recovery, and, on the other hand, that a rapid convalescence does not exclude the possibility of a later development of tuberculosis.

Sudden death in the course of acute pleurisy is not unknown, and seems to be dependent upon thrombosis of the right side of the heart.

Pericarditis is a serious complication.

TREATMENT.—The general rules applicable to the pyrexial state should be observed. In the early stage the patient's chief complaint is of pain, which is usually severe. A hypodermic injection of morphia, repeated if necessary, and followed by a few doses of Dover's powder, affords a safe and efficient means of relief. The abstraction of blood by leeching, wet cupping, or venesection, is very rarely necessary. The respiratory movements being intensely painful, much relief will be obtained by tightly bandaging the side, or by the application of strips of plaster, after the manner recommended by F. T. Roberts. Poultices of linseed and mustard, or hot fomentations combined with laudanum, are often grateful to the patient. The bowels should be moderately opened and a febrifuge mixture administered. The diet should be liquid. The above measures usually



suffice until the effusion has reached its height. In a large proportion of cases the effusion is spontaneously absorbed, and active treatment is both unnecessary and useless. The bowels should be kept open without active purgation, and a mixture containing acetate of potash, spirit of nitrous ether, and digitalis, will assist excretion by the skin and kidneys. The value of the time-honoured blister of cantharides or iodine is very doubtful. Our chief concern at this stage is to watch for the normal recession of the tide of effusion. As the pyrexia abates the writer is in the habit of substituting a mixture containing 2 or 3 grains of iodide of potassium, combined with 3 or 4 grains of the citrate of iron and ammonia *pro dosi* for the diaphoretic and diuretic mixture already mentioned. If the effusion soon begins to show signs of subsidence a general tonic line of treatment should be early undertaken, bearing in mind the dangers of a lingering convalescence or of the development of tuberculosis. If the effusion persists at its highest level the question of more active treatment must be considered. We have to choose between such measures as active purgation, blistering, and the limitation of fluids on the one hand, and aspiration on the other. The writer has seen little success from the former methods, while the results of aspiration are often most satisfactory. Osler, whose opinion must carry great weight, states that he has seen large effusions disappear rapidly under a dry diet and full doses of Epsom salts. These methods may have a trial, but if no signs of absorption appear in the course of the third week aspiration should be performed if there be no contra-indications. The question arises whether we are justified in waiting so long before resorting to this expedient. It is probable that we are justified for the following reasons:—In the course of the first two weeks spontaneous absorption is always probable. The dyspnoea consequent on the exudation is rarely so urgent as to demand relief at an earlier stage, but urgent dyspnoea should be regarded as an indication for aspiration at any stage of the disease. It is remarkable, however, how soon patients get accustomed to the effusion and how little they suffer from dyspnoea, so long as they make little or no exertion. Further, early aspiration seems to increase the tendency to a fresh accumulation of fluid, and it does not seem to prevent deformity of the chest. One of the worst cases of falling in of the side ever observed by the writer occurred in a child in whom, owing to somewhat urgent dyspnoea, it was thought advisable to perform aspiration about the seventh or eighth day of the disease. Large effusions, as a rule, absorb tardily, and in most of these cases aspiration will be required. Aspiration is also indicated in the rare cases in which the effusion is bilateral. As regards the proportion of cases in which aspiration will be called for no rule can be laid down. In one series in the writer's practice aspiration was performed in thirteen out of thirty-nine cases. He has never had reason to regret the performance of the operation, but has sometimes regretted that for some reason or another the operation was postponed or omitted. The effect of aspiration varies. In exceptional cases it is followed by rapid convalescence, and no further treatment is required. The writer can recall a case of a young girl whose left pleura had been allowed to remain full of fluid for eight or ten weeks, and who rapidly convalesced after a single aspiration. Such results are, however, exceptional. Much more often the fluid reaccumulates, and the operation has to be repeated. The effect of aspiration upon the temperature is variable. In four of the thirteen cases already mentioned aspiration was followed by a marked fall in temperature. In several cases aspiration had no effect upon the temperature, and in one case the pyrexia seemed to



be aggravated by the operation. The idea that persistent pyrexia contra-indicates aspiration seems to be without foundation.

The management of the convalescent stage of pleurisy is a matter of the utmost consequence, to which hardly sufficient attention is usually paid. We shall first consider the management of this stage in cases which pursue a fairly normal and favourable course, and, secondly, in those troublesome and difficult cases of recurring and persistent effusion. In the former cases a definitely tonic line of treatment should be early adopted. When the pyrexia has disappeared or becomes trifling, the patient should leave his bed, take gentle exercise at first indoors, and before long in the fresh air, and be instructed to breathe deeply and use the dumb-bells. A change of air—preferably to upland air—and gentle daily walking exercise on moderate inclines will often have the happiest results in these cases. Singing may be practised with advantage. The diet should be as liberal as the state of the digestion will permit. The possibility of subsequent phthisis is always to be borne in mind, and a winter at one of the Alpine stations will sometimes be advisable. It is reasonable to hope that by such measures the proportion of these cases which ultimately become phthisical may be materially reduced. Much more difficult is the management of those cases, by no means rare, where in spite of repeated aspiration the fluid obstinately reaccumulates, and no real progress is made. Aspiration should have a fair trial, but no rule can be laid down as to how often it should be repeated. The writer is averse to very frequent repetition of the operation, except as a palliative measure in obviously incurable cases. It seems to be generally agreed that free incision and drainage is not to be advised in these cases, and that injections of astringents or antiseptics are useless. The writer has no experience of these methods. Perhaps the best course to pursue in these unpromising cases is to adopt some approximation to the tonic and hygienic line of treatment advised for the more favourable class. At best the outlook is gloomy. Too often the heart becomes dilated, dropsy sets in, or else an intractable empyema is set up, or in some other way the general health is ruined. Lardaceous disease sometimes marks the final stage. Probably a large proportion of these cases are tubercular.

In dealing with any case of pulmonary disease the fact of an antecedent pleurisy should always be allowed considerable weight. It suggests caution in prognosis and a careful search for evidence of tuberculosis. It should not be forgotten in connection with life assurance forecasts.

(For a discussion of the surgical aspects of aspiration, see "PARACENTESIS THORACIS," p. 403.)

#### PLEURISY IN CHILDREN

"Pleurisy in children," says Henoeh, "differs in no essential particular from the same disease in later life." This is true, there being no features of the disease peculiar to any period of life, yet pleurisy in the child requires special attention. The difficulties of diagnosis are greater in the child than in the adult, and misleading signs and symptoms are more common. We shall briefly summarise the more important points.

(a) *Mode of Onset*.—Vomiting, epileptiform convulsions, and other symptoms of cerebral irritation, are commoner in the child than in the adult, but, on the other hand, an insidious onset is very common in the child, and the disease can be recognised only by physical examination.

(b) *Nature of the Effusion*.—The effusion is more often purulent in the child than in the adult. Goodheart found pus in 78 out of 149 cases of



pleural effusion in childhood. It is about an even chance whether we shall find pus in any case of acute pleurisy with effusion occurring in a child. Loculation of the fluid is common.

(c) *Physical Signs*.—The peculiarities of the physical signs of pleurisy in the child depend upon the facts that at this age the chest walls are more yielding, and the lung tissue more elastic and less easily compressed than in the adult. Deep percussion will often yield resonance, even in the presence of a large effusion. Light percussion is more satisfactory. Skodaic resonance is well marked. Alterations in the tactile fremitus are less trustworthy in the child than in the adult.

Bronchial breathing is the rule in childhood, either early in the case or lasting throughout the entire effusion stage. It is often but not always weak and “distant” in quality. Sometimes the breathing has the normal vesicular character, but is less distinct than in health. “Good though deficient vesicular murmur,” says Goodheart, “may be present all over the side which is full of fluid, and unless this is remembered there is likely to be a mistake in diagnosis.” In certain cases of pleurisy in children the signs, especially in the mammary region, may closely simulate those of cavity. The breathing is hollow, and there are adventitious sounds not easily distinguishable from gurgling râles. In such cases the general history of the case, the examination of the sputum, the position of the heart, and the result of puncture, may be relied upon to establish the diagnosis. The exaggeration of the breath sounds on the healthy side is marked.

(d) *General Considerations*.—Pleurisy is more often a sequel of the acute infective diseases in the child than in the adult. Pericarditis is said to be commoner in early life than at a later period. The course and results of pleurisy do not present any striking peculiarities in childhood. Rapid absorption is common, but so also is a lingering and tedious convalescence. Marked deformity of the chest may remain after an attack, but often undergoes much subsequent amelioration. In empyema the prospects of cure by simple aspiration are much better in the child than in the adult, but incision and drainage will usually be necessary.

VARIETIES OF PLEURISY.—No satisfactory classification of pleurisies is at present possible. Most of the usual varieties have been incidentally alluded to in the course of the foregoing description. The subject can only be very briefly considered here.

Most of the classifications which have been adopted involve unverifiable hypotheses, or seek to establish distinctions which a fuller knowledge may show to be invalid. Enough has been said to show that the differentiation of tubercular pleurisy from other varieties, which is the great practical desideratum, cannot be established with certainty either by clinical or pathological evidence. “Latent” or “quiet” pleurisy has no claim to be regarded as a separate entity, nor can “diaphragmatic pleurisy” be erected into a distinct variety. It is very questionable if pleurisy ever remains limited to the reflection of the pleura upon the diaphragm (see vol. ii. p. 508, “Diaphragm”). Three types of pleurisy remain for consideration which may fairly demand separate consideration, viz. dry pleurisy, and chronic pleurisy and empyema.

*Dry Pleurisy*.—In this condition the pleural surface is covered in part with one or more layers of lymph, and effusion does not take place. The symptoms are pain in the side, slight pyrexia, which may, however, be absent, and some degree of general malaise. Friction is heard on examination of the chest. It is commonly posterior or antero-lateral in situation,



usually in the neighbourhood of the base of the lung, and is most often quite limited in extent, but in exceptional cases may be heard almost over the entire side of the chest. The course of these cases is often tedious, but ultimately adhesions usually form between the opposing pleural surfaces, and recovery takes place.

The same doubt overhangs the ætiology and significance of these cases which appertains to the subject of acute pleurisy. Whether a "dry" pleurisy is ever the result of simple chill, whether it ever arises in an individual otherwise quite healthy, whether the presence of this condition warrants a suspicion of tuberculosis, whether the after-history of these cases shows that their ultimate course is unfavourable—all these questions are at present unsettled. The fact that in almost every patient dying of a chronic malady some adhesions of the pleuræ are found, points to the conclusion that this membrane readily undergoes plastic exudation in patients whose general condition is bad, or who are the subjects of some constitutional malady. To infer that the pleural adhesions so constantly found in the deadhouse are always, or even in the majority of cases, tubercular, seems a large and unverifiable assumption. Direct proof of their tubercular nature is usually wanting. On the other hand, it seems doubtful whether a "dry" pleurisy ever arises as the result of simple chill in an individual otherwise quite healthy, and a pleurisy of this type should be watched with as much, or even greater care than an ordinary acute pleurisy. Where friction is limited to the apical region the suspicion of tuberculosis will be much strengthened.

The treatment consists in the relief of pain, general tonic measures, and watchfulness regarding possible developments. The subject of "dry" pleurisy should, if possible, shun indoor occupations, and all depressing conditions of life and work should be reduced to a minimum. The sputum, if obtainable, and the urine should be carefully examined. It must not be forgotten that in exceptional cases "dry" pleurisy takes on an acute development, and effusion may suddenly set in.

*Chronic Pleurisy.*—This subject has been already incidentally considered. The main facts may be here conveniently summarised. Chronic pleurisy may be a sequel of the acute or subacute affection, or the attack may be chronic throughout. It may be attended by moderate or large effusion, or may belong to the type of the so-called "dry" pleurisy. The so-called "latent" or "quiet" pleurisy already described, in which a large effusion collects without definite symptoms, is one variety of chronic pleurisy with effusion. In another class of cases an ordinary attack of acute or subacute pleurisy is characterised by persistence of the effusion, or by its repeated recurrence after aspiration. A large proportion of these cases are probably tubercular. In a third type of case the exudation is plastic, more or less extensive adhesion of the pleural surfaces takes place, and the function of the lung is in some degree impaired. In very chronic cases thickening of the pleura to an extreme degree may occur, the newly-formed tissue is often tough and unyielding in consistency, and sometimes it undergoes calcareous change. The lungs may undergo cirrhotic change. The general course and symptoms of chronic pleurisy present great variations. Amongst the most constant features are shortness of breath, cough, progressive debility, slight cyanosis, and diarrhœa. Pain is usually present, but may be slight, and may not be mentioned by the patient until inquired for by the physician. At a late stage of some of these cases the symptoms of dilatation of the heart may complicate those proper to pleurisy.

The physical signs of chronic pleurisy depend upon the amount of the



effusion, the degree of collapse of the lung on the affected side, and the consecutive changes in the chest wall. In some cases retraction of the side is a prominent feature, in others there is no retraction. Dulness on percussion, impairment of tactile fremitus, and feebleness of the respiratory murmur are usually present, but in very varying degree. In some cases friction is the only physical sign.

The prognosis is unfavourable as regards complete *restitutio ad integrum*, but some of the less grave cases preserve a fair degree of health and fitness for work for long periods. It is not well to take a grave view of slight cases of chronic pleurisy, but the possibility of tuberculosis must always be borne in mind. Cases of persistently recurring large effusions are very unfavourable. The treatment has been already considered (see p. 390).

### EMPYEMA

The bacteriology of empyema has been already considered. The organisms most often found are the pneumococcus, the streptococcus, the staphylococcus, and various bacteria of putrefaction. The bacillus tuberculosis is, on the whole, rare. There is a growing consensus of opinion amongst bacteriologists that a pneumococcus empyema is the most favourable variety, and a streptococcus empyema the least favourable. The significance of the presence of the bacillus tuberculosis must be estimated on general principles.

*Ætiology.*—In a large proportion of empyemata the effusion is purulent from the onset, especially in children, and due either to some intensity of the morbid process or to the weak resisting power of the individual. The idea that serous pleurisy often become purulent as the result of repeated aspiration rests on no secure evidence. If, however, on a first aspiration the fluid is somewhat turbid in appearance and is found to be rich in cellular elements, empyema will probably result. These cases are from the outset on the way to become purulent.

Pneumonia is one of the most important causes of empyema, which may either accompany or succeed the pneumonic attack. In the former case the clinical course and the physical signs may present some puzzling features until the diagnosis is made out. The attack may set in with the ordinary signs and symptoms, and for a few days no special features may be present. But the crisis is delayed, the expected improvement does not take place, the looked-for crepitus redux does not appear, and gradually unequivocal signs of fluid in the pleura make their appearance. Puncture should be early practised in these cases as a prompt diagnosis is imperative.

The pleurisy connected with scarlet fever and typhoid is often purulent.

Empyema may result from disease of bone, wounds, and injuries of the thorax, malignant disease of the lungs or the œsophagus, rupture of an abscess or a tubercular vomica into the pleural cavity.

Tuberculous pleurisy is sometimes purulent, but is much more frequently sero-fibrinous.

Empyema may occur in association with septicæmia.

*Pathology.*—The effusion presents all gradations from a slightly turbid sero-pus to a thick, inspissated lumpy mass, the former condition being characteristic of recent, the latter of chronic cases. Often the effusion is of the consistence of cream. Fœtor is unusual, but may be found in traumatic cases, and in association with gangrene of the lungs or pleuræ.

Thickening of the pleura is usual, and may attain an extreme degree in very chronic cases. The fibrinous exudation becomes organised into granulation, and ultimately into connective tissue, and adhesions are formed. Loculation is common. The pus may become impregnated with calcareous salts, and calcareous matter may be deposited in thick layers upon the pleural surface.



*Diagnosis.*—There is only one means of diagnosing empyema, viz. by puncture and withdrawal of some of the fluid. Often, no doubt, the presence of pus may be correctly surmised, but neither by signs nor symptoms can the surmise be translated into certainty. The physical signs of empyema and those of serous effusion are identical and have been already fully considered. Nor will the symptoms suffice to afford a secure diagnosis. The writer has seen marked hectic fever, profuse perspiration, and wasting in cases where on aspiration the fluid proved to be limpid serum, and, on the other hand, has found pus where there was little or no pyrexia or other active symptom. Edema of the side and clubbing of the fingers are equally fallacious. These signs point to the chronicity of the case, but shed no certain light upon the character of the effusion. The history will often give a hint, but it must be reaffirmed that until some of the fluid has been withdrawn the diagnosis remains simply a more or less probable guess. In making the exploratory puncture some precautions are necessary. The exploring syringe or trocar and canula should not be too fine, otherwise the pus will not flow through it. The thickening of the pleura may be extreme, and great difficulty may arise in gaining access to the pleural cavity. Further, loculation is common, and the pus may be missed. This error may usually be avoided by the operator taking care to insert the instrument at a point where the signs of the presence of fluid are unequivocal.

*Treatment.*—This is purely surgical (see “Surgical Section,” p. 404).

#### HYDROTHORAX

Dropsy of the pleural cavity or serous effusion not due to primary changes in the pleura is a common condition, and is not unfrequently overlooked. It is common in heart disease, Bright’s disease, or other chronic renal malady, and may be found in connection with aneurysm, thoracic tumours, chronic hepatic disease, and anæmia.

The nature of the effusion has been already considered. It is usually distinguishable from inflammatory or bacterial effusion by its lower specific gravity, its relative pooriness in corpuscular elements, and its tardiness in coagulating.

The physical signs are not to be distinguished from those of ordinary pleural effusion, but friction is not present, and as the effusion is often double the heart may remain in its normal position. It is remarkable, however, how often the effusion is unilateral in cases where a bilateral effusion might be expected. A unilateral effusion is quite common in cardiac cases, perhaps more common than a double effusion. A very usual condition in these cases is to find a considerable effusion on one side, with a slight and barely recognisable effusion on the other. In renal disease the effusion is more often on both sides. Passive effusion into one or both pleural cavities is a very usual explanation of the aggravation of symptoms in a cardiac case, and should always be carefully borne in mind. In such patients, however, so long as they are confined to bed and making little or no exertion, a considerable degree of effusion may take place into one or both pleural cavities without any marked increase of dyspnoea.

The treatment of hydrothorax, apart from general medical measures, is aspiration, which should always be practised where there are any active symptoms. The prospects of spontaneous absorption are rarely good. They are, perhaps, best in cardiac cases.



## HÆMOTHORAX

An effusion of pure blood into the pleura probably occurs only as the result of trauma, or the rupture of an aneurysm, but effusions of more or less sanguineous serum (more rarely pus) are not very infrequent. The causes of this condition have been already indicated, viz. tuberculosis malignant disease, the exanthemata, hæmophilia, scurvy, cirrhosis of the liver, and possibly inflammatory changes in profoundly debilitated subjects.

The physical signs do not present anything distinctive, but the associated conditions may give rise to a somewhat complicated clinical picture.

The chief interest of these cases lies in the prognostic significance of sanguineous effusion. This varies within wide limits, according to the ætiology, but for obvious reasons the prognosis is rarely favourable. A satisfactory issue, though not often to be anticipated, is by no means unknown. The writer has known comparative recovery with restoration of health for several years to follow a case of large sanguineous effusion in a young gentleman who was probably tubercular.

As regards treatment, aspiration is not to be lightly undertaken, but the propriety of the operation may be considered in cases where the amount of the fluid is causing serious inconvenience. Several cases are on record where the operation has been followed by recovery, but it may be attended by considerable risks. The presumption is, on the whole, against this procedure in hæmothorax.

## PNEUMOTHORAX

Air in the pleural cavity is almost always associated with fluid—rarely serum, usually pus. That air is sometimes effused into the pleural cavity and subsequently absorbed without appreciable exudation is probable, but the clinical importance of these cases is slight. Practically pneumothorax and pyo-pneumothorax are almost convertible terms. In a large majority of cases pneumothorax is a consequence of tuberculosis.

*Ætiology.*—It is probable that pneumothorax is never a primary affection. Laennec believed the contrary, and held that it might arise by the exhalation of air from the pleura or by the decomposition of fluids in the pleural cavity. These views have not been confirmed by subsequent observers. Whether bacteriology will throw much new light on the ætiology of pneumothorax is still doubtful. E. Levy describes a bacillus which he has found in the exudate of pneumothorax, and to which he is disposed to attach importance. It is a short, thick bacillus with rounded ends, anaerobic, non-motile, and readily stained by aniline dyes. It occurs frequently in pairs, but often, also, in long chains or threads. Spore-formation was not observed to occur. At a temperature of 37° C. the growth of this bacillus proceeds actively with a remarkable evolution of gas.

The two chief causes of pneumothorax are (1) the breaking down of a subpleural nodule in the course of pulmonary tuberculosis, and (2) the discharge of an empyema through the lung or the chest wall. Of remaining causes trauma and pulmonary gangrene are the least infrequent. Emphysema has been educed as a cause in a few instances, and inasmuch as bullæ containing air are common enough on the pleural surface in this disease, there is no antecedent improbability that pneumothorax might result. If these bullæ rupture it seems likely that the air is often reabsorbed, and that pneumothorax does not as a rule ensue. In one case in the writer's



practice pneumothorax was the indirect result of gastric ulcer, an abscess forming behind the stomach, which penetrated to the pleural cavity through the diaphragm, and ultimately discharged through the lung. Abscess of the lung, cancer of the lung, "apoplexy" of the lung, have been regarded as causes of pneumothorax. Such cases must be of extreme infrequency. The writer's experience is entirely in accord with the statement of Douglas Powell—"excluding gunshot wounds, the discharge of empyemata and gangrene of the lung, it is infinitely rare for pneumothorax to occur from any other cause than the breaking down of subpleural consolidations in phthisis."

The admission of air to the pleural cavity in consequence of the aspiration of an empyema is an occasional cause of pneumothorax.

As regards the relative frequency of the various causes of pneumothorax pulmonary tuberculosis easily takes the first place. Saussier, whose statistics are quoted by Fraentzel, found pulmonary tuberculosis in 81 out of 131 cases of pneumothorax. These figures, as Fraentzel justly remarks, "evidently give a too small comparative proportion to pulmonary consumption." He regards this cause as present in about 14 out of every 15 cases. Walshe puts the proportion of cases coming under this head at 90 per cent. Empyema is a somewhat frequent cause of pneumothorax. Saussier found this cause present in 29 of his 131 cases. The other causes, excepting perhaps gangrene of the lung, are quite rare.

As regards the proportion of cases of pulmonary tuberculosis in which pneumothorax occurs, we find divergent views. Neil put it at 13 per cent, Williams at 10 per cent, West and Powell at 5 per cent. The writer's experience tends to corroborate the last figure.

Authorities differ as to which side is oftener affected, but the point is immaterial.

*Pathology.*—Most observers incline to the opinion that pneumothorax is most apt to occur in acute cases of pulmonary tuberculosis, especially in the acute caseating form, but Fraentzel holds the contrary view, and asserts that in his experience pneumothorax has arisen most often in chronic cases, and usually at a very advanced stage of the disease. This has also been the experience of the writer. Acute miliary tuberculosis and fibroid tuberculosis seem to have little tendency to cause pneumothorax. Violent fits of coughing and straining at stool seem to be the usual exciting causes of pneumothorax in tubercular cases. The tubercular masses break down near the surface of the lung, necrosis of the pleura occurs, and pneumothorax follows. If it were not for the formation of protecting pleural adhesions, pneumothorax would be a common, instead of a rare phase of pulmonary tuberculosis.

There may be one or several openings, and the opening or openings may be large or small, patent and direct, or oblique and valvular. As regards the seat of the perforation, Williams found it in the upper lobe in seventeen instances, in the middle lobe in four, and in the lower lobe in sixteen, in the tubes between the upper and middle lobes in one. Walshe was of opinion that "the pleura commonly gives way postero-laterally in the area comprised between the third and sixth ribs." West's figures give a large preponderance to the upper lobe.

The mode in which empyema or pulmonary gangrene gives rise to pneumothorax is too obvious to demand any detailed description.

The gas effused into the pleural cavity is found to have approximately the characters of expired air. J. Lasch states, on the authority of some unnamed observers, that by estimating the amount of oxygen and carbon dioxide in this gas, we can determine whether or not there is a free communication between the bronchi and the pleural cavity. If a free opening exists, the proportion of oxygen is over 10 per cent, that of carbon dioxide not exceeding 5 per cent, while if no such opening exists the proportion of oxygen is under 10 per cent, that of carbon dioxide over 10 per cent.

Douglas Powell estimated the state of the intra-pleural pressure in sixteen cases of pneumothorax. He found the pressure *nil* in four cases, and in twelve cases the pressure varied from  $1\frac{3}{4}$  to 7 inches of water.



CLINICAL HISTORY.—*Mode of Onset.*—In a considerable proportion of cases the onset is sudden and well marked, and symptoms of severe shock are present. The patient feels a severe pain in the chest, he has the sensation “as if something had given way,” dyspnoea becomes urgent, the pulse is much accelerated, and a clammy perspiration breaks out. Cyanosis is often a marked feature. The decubitus of the patient varies. Cough and expectoration sometimes cease temporarily.

It is by no means rare, however, to find these symptoms absent or ill marked. As Fraentzel truly observes, pneumothorax often comes on in advanced phthisis “without any particular complaint on the part of the patient, and without any decided aggravation of the previously existing dyspnoea; and it is only on a closer examination that we discover the presence of an accumulation of air in the pleural cavity.” In cases where pneumothorax supervenes upon empyema the first indication of its presence may be the ejection of a large quantity of pus from the mouth. It is probable that the acuteness of the symptoms of onset has some relation to the functional activity of the lung on the affected side at the moment when perforation of the pleura occurs. If the lung be comparatively sound the symptoms will probably be urgent, while if it be extensively damaged by consolidation, softening, and excavation, the symptoms will tend to be more latent. According to Douglas Powell, the symptoms of pneumothorax “may be most closely simulated in an attack of acute pulmonary congestion supervening upon already advanced disease.”

*Progress of the Case.*—The patient may die within a few hours from shock, but this is rare. Usually he rallies, unless he is already in the last stages of exhaustion consequent on advanced pulmonary phthisis, and his condition may either show little change as the result of the new phase of his malady, or he may gradually sink, or in exceptional cases the occurrence of pneumothorax may tend to retard the progress of the pre-existing tuberculosis. Recovery is possible, especially in cases consequent on empyema (see “Prognosis”).

The temperature in pneumothorax shows much variation. In cases where the onset of the condition is attended by much shock, there is usually, as might be expected, a sudden fall followed by a sharp rise. The course of the temperature is most often similar to what is found in tuberculosis.

*Physical Signs.*—These are usually definite and characteristic.

(a) *Inspection.*—The affected side is distended and immobile. The intercostal spaces are flattened. The movements of the sound side are exaggerated. The impulse of the heart may be seen to be displaced towards the sound side. The shoulder on the affected side is raised.

(b) *Palpation.*—Vocal fremitus is abolished. The liver and spleen may be felt to be displaced downwards. This is common in pneumothorax, while it is rare in pleural effusion.

(c) *Percussion.*—The percussion note is hyper-resonant or tympanitic, unless the distension of the side is excessive, when the so-called “tympanitic dulness” may be present. Cardiac dulness is often obliterated.

(d) *Auscultation.*—The breath sounds are weak or absent. In some cases they are faintly amphoric. Vocal resonance is diminished.

Certain special auscultatory signs are found in pneumothorax, and are of much assistance in diagnosis. These are: (1) Metallic tinkling; (2) The bell sound (*bruit d'airain*); and (3) Succussion splash. (1) Metallic tinkling was compared by Laennec to the sound produced when a metallic, glass, or porcelain cup is struck by a pin. He explained it as caused by



drops of fluid falling in an air-filled cavity. Its mode of production is doubtful. (2) The bell sound, or *bruit d'airain*, is a highly characteristic chiming sound, produced over a pneumothorax by placing one coin upon the chest wall and striking it with a similar coin. (3) Succussion splash. If the patient is suddenly shaken, while the observer listens through the stethoscope, a splashing sound is often clearly audible.

Metallic tinkling, the bell sound, and the succussion splash, simply indicate that fluid is present in a large air-containing cavity, and are not pathognomonic of pneumothorax. They may arise in connection with large phthisical cavities, or a distended stomach, or a distended colon.

The physical signs of pneumothorax may be modified by: (1) The state of the lung on the affected side, *e.g.* by consolidation or excavation; (2) The state of the lung on the sound side. This lung is frequently the seat of disease, and the physical signs will vary accordingly; (3) The amount of fluid present in the affected side.

*Diagnosis.*—This is usually obvious. Attention to the history and the physical signs rarely leaves much room for uncertainty. Practically, almost the only condition likely to give rise to difficulty is a large, thin-walled, pulmonary vomica; but in such cases the heart is not displaced towards the sound side, the side is flattened rather than distended, the bell sound will probably be absent, and the distribution of the physical signs is different from that which prevails in pneumothorax.

*Prognosis.*—This must have regard both to the antecedent condition and to the special dangers arising from the occurrence of pneumothorax. Two questions present themselves for discussion: (1) What is the prognostic significance of the occurrence of pneumothorax in a case of chronic pulmonary phthisis? and (2) What importance must be attached to pneumothorax supervening upon empyema?

As regards the former question, it may be answered that pneumothorax occurring in the course of pulmonary phthisis usually precipitates the fatal issue. Douglas Powell found the average duration of life in thirty-nine cases of this kind to be only twenty-seven days. In the most favourable case the duration of life was twelve months. Weil found that out of forty-five cases about one-half were dead within a month, while some survived for years.

In rare cases the occurrence of pneumothorax seems to retard the progress of pulmonary tuberculosis. The writer has seen one striking case of a young man who decidedly improved after pneumothorax had occurred in the course of chronic phthisis. The patient regained a considerable degree of health, but was subsequently lost sight of.

As regards the second question, pneumothorax supervening upon empyema sometimes—it is difficult to say in what proportion of cases—takes a favourable course. In two recent cases in the writer's practice recovery took place. One of the patients was found to be in good health three years subsequent to the occurrence of pneumothorax.

According to Fraentzel, pneumothorax occurring in association with gangrene of the lung is usually fatal within twenty days.

*Treatment.*—If shock be pronounced at the outset appropriate measures—such as morphia or ether by hypodermic administration, and the exhibition of alcohol—must be employed. If there be evidence of excessive intra-thoracic pressure a fine trocar should be introduced, and some of the contained air allowed to escape. It must not be forgotten that this procedure is attended by the danger that the diminution of the intra-thoracic pressure may cause the reopening of the perforation of the pulmonary cavity in cases where



pneumothorax has arisen in advanced phthisis. Hence great caution should be used, and aspiration is not to be employed. If, as is almost invariable, fluid as well as air be present in the pleural cavity, some of it should be withdrawn and examined. Dr. Finlay is of opinion that if the fluid be serous "the general condition of the patient will be no worse than if air alone were present; probably, indeed, better, as the pressure exerted on the lung may tend to check the progress of disease in it, and will promote the effectual sealing up of the perforation. If the liquid be foetid pus nothing but harm can come from letting it remain in the pleura, and it ought to be evacuated at once." The same authority, referring to cases where the fluid is purulent but not foetid, advises that, "if the pneumothorax have resulted from rupture of an empyema into the lung, the chest should be freely opened and drained, and the same would hold good if the empyema had ruptured through the chest wall, the opening which nature makes not being, as a rule, sufficient for free drainage. And even in the case of pneumothorax of tuberculous origin a consideration of general principles dictates the free evacuation of the pus." As to the propriety of leaving a serous effusion in pneumothorax undisturbed there can be no second opinion. Such cases are, however, of little practical importance. Foetid pus should, it is clear, be promptly evacuated by incision and drainage. The treatment of pneumothorax consequent upon empyema is not quite so certain. There is much to be said for the advisability of freely opening the chest in the hope that the pus will thus easily drain from the most dependent part, and that the broncho-pleural fistula will close, but success will sometimes attend an expectant policy. Two of the best cases of recovery which have occurred in the writer's practice were cases belonging to this class in which for various reasons no operative measures were undertaken. Operation in cases of pneumothorax consequent upon the rupture of a vomica into the pleural cavity is of most doubtful expediency. Many of these cases are already hopeless. The patients, as a rule, are ill fitted to bear operative interference. There is always the bare chance that the occurrence of pneumothorax may not alter the patient's condition for the worse. In view of these considerations, the safer rule would appear to be to recommend operation only when the urgency of the symptoms or the special indications of the individual case would appear to indicate its advisability.

The general treatment of pneumothorax in cases which survive the early dangers of the condition must be on tonic and hygienic lines. In rare cases success may be achieved when little expected.

**MALIGNANT DISEASE OF THE PLEURA.**—This subject will be considered in detail in the surgical section. A brief reference to it must suffice in this place. Primary malignant growths are rare in the pleura, but may occur in the form of endothelioma or sarcoma. Secondary deposits are much more frequent. In such cases the seat of the primary affection is usually the lungs, mediastinum, or mamma.

The diagnosis may present considerable difficulties until the case has made progress. The onset is insidious, pyrexia is slight, pain is occasionally present and may be severe, but this is not invariable, shortness of breath and cough are usual, and as time goes on progressive emaciation and debility will probably appear. The physical signs are for a time simply those of effusion. Examination of the fluid may give important indications. It is usually hæmorrhagic, and may contain cancer cells (see "Hæmorrhagic Effusion," p. 396). The fluid may, however, be simply serous, and present no distinctive features. At a later stage signs of pressure will probably appear with displacement of organs.



The duration of these cases is from a few weeks to from three to six months.

The treatment must be on general lines. Paracentesis should not be performed unless it is urgently indicated by pressure symptoms. (See "SURGICAL DISEASES OF THE PLEURA," *infra*; and "MEDIASTINUM," vol. vii. p. 365 *et seq.*).

*Lymphatics of the Pleura.*—As in other serous membranes, lymphatic vessels are abundant in and beneath the pleura. They are found in large numbers in the thin layer of areolar tissue underlying the serous covering. "These vessels communicate on the one side by means of stomata with the pleural cavity, and on the other with a network of similar vessels in the interalveolar septa of the lungs" (Quain).

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INJURIES OF THE PLEURA.—Severe contusion of the chest, without fracture or external wound, may cause rupture of both pleural layers, and be followed by surgical emphysema (*q.v.*), pneumothorax, hæmothorax, or inflammation of the pleura with effusion. Such an injury will probably be associated with graver damage to the lung, and the symptoms and treatment may, in some measure, be influenced thereby. As the air is mechanically freed from germs in its passage through the lungs it enters the pleural cavity quite pure, and accordingly such injuries, in nearly every case, may be expected to run an aseptic course.

*Emphysema* of the cellular tissue may be left to Nature, as the air becomes absorbed in about a week. It is only when the air is widely diffused and its pressure embarrassing, that it may be dealt with by multiple punctures with a fine trocar.

*Pneumothorax*, thus arising, may also be watched, unless the dyspnœa be extreme, when a fine trocar and cannula may be employed for the release of the air. It is not desirable to use an aspirator in either of these conditions.

*Hæmothorax*, in like manner, should not be interfered with unless the dyspnœa be distressing; and if the aspirator be used for the evacuation of the blood, care should be taken not to draw fresh blood by undue suction.



The mechanical assistance of the air and blood in the cavity, with consequent compression and temporary shrinkage of the lung, is favourable to the closure of the rent in the visceral pleura, hence the excuse for non-operative measures, save in extreme dyspnœa.

Lacerations of one or both pleural layers may occur in simple fractures of the ribs or sternum, particularly in those due to direct violence, and the same principles of treatment apply.

*Penetrating Wounds.*—Wounds of various kinds, including gunshots and compound fractures, admitting air from without to the pleural sac are regarded as penetrating, the parietal layer of the membrane being considered the boundary. Apart from the associated visceral damage, these injuries derive their gravity from septic matters carried in from the surface, scraps of clothing and metallic bodies lodged in the sac. It should be remembered that the pleuræ may be wounded through the abdomen or at the root of the neck.

*Traumatopnœa*, or breathing through the wound, is a sign of penetration, but it does not necessarily imply wound of the visceral pleura.

In obscure cases it is unwise to probe small wounds lest penetration be thereby caused. It is better practice to enlarge the surface wound and examine it with the finger, if it be desirable to ascertain its depth, or to deal effectively with intercostal hæmorrhage.

Small incised wounds, after thorough antiseptic cleansing, may be closed at once, and if sutures are used, they should not include the parietal layer. Larger wounds, and all punctured and gunshot wounds, after similar careful disinfection, should be covered with a copious absorbent antiseptic dressing and binder. It is better not to close them completely, so that drainage may be secured. Many such heal without inflammation. Even gunshot wounds with lodged bullets often do well, as the experience of the South African war abundantly illustrates; and it further teaches, that prolonged search by extensive operations for these projectiles is not the best course to adopt. Lodged foreign substances, if readily accessible, should of course be extracted, either through the wound, which may be enlarged for this purpose, or through an independent incision; metallic bodies being located by the X-ray apparatus when available.

In the later management of these cases should temperature, pulse, and dulness indicate inflammatory effusion, the character of this may be estimated by the exploring syringe. If purulent, arrangements must be made for free drainage, either by enlarging the wound, or if this be in an unfavourable situation, by an independent opening, according to the principles laid down in the section on empyema.

**TUMOURS OF THE PLEURA.**—Primary neoplasms are rare. Secondary growths arising by extension of tumours from the lung, mediastinum, breast, or adjacent bones are not uncommon.

Primary Tumours.	Secondary Tumours.
1. Fibroma.	1. Sarcoma.
2. Sarcoma.	2. Cancer.
3. Endothelial cancer.	

*Fibrous tumours* have been described by Kingston Fowler and Steven forming broad thickenings of the pleura with dense adhesions to the lung. Their structure is laminated, enclosing small cysts, very dense and glistening on section, and of a pink colour. They are incapable of surgical extirpation.

*Sarcomata* in all varieties occur, but the round-celled is the commonest.



*Endothelial tumours*, which are microscopically indistinguishable from true cancers, are supposed to spring from the pleural endothelium, or from the original surface epithelium of the embryo. These either form whitish nodules scattered over the parietal layer, or broad, flat, continuous infiltrations covering the entire membrane.

*Diagnosis*.—The early symptoms are obscure: shortness of breath, loss of strength and weight, and coughing. There may be slight fever, irregular vomiting, and albuminuria. Pain may be wholly absent, slight, or persistently severe. At a later stage dark or bloody effusion will probably be found, and this may be the first sign to suggest the diagnosis. The fluid may be microscopically examined for cell elements, for it must be borne in mind that hæmorrhagic effusion may occur in many other states, like tuberculosis, scurvy, nephritis, the exanthemata, and hæmophilia. After aspiration in tumour cases the dulness, diminished fremitus, and resonance may remain. Further, the displacement of the heart or diaphragm is remarkably slight, and there are usually no signs of pressure on the vessels, trachea, or œsophagus. The chest may even be retracted in some cases.

*Prognosis* is uniformly unfavourable. Most sufferers die in from two to six months, in rare instances they linger for one or two years.

*Treatment*.—Extirpation is impossible. Even aspiration should not be resorted to very frequently, but only used when the dyspnoea demands.

#### EXPLORATION OF THE PLEURA

*Paracentesis Thoracis — Thoracentesis — Aspiration of the Pleura*.—Aspiration or tapping of the pleura may be required in hydrothorax, hæmothorax, chylothorax, pneumothorax, tubercular and malignant effusions, ordinary serous pleurisy, and empyema. It may be regarded as exploratory or curative. The reader is referred to the preceding articles for the indications as to its employment in these conditions.

The operation may be performed with a trocar and cannula, or the cannula may be furnished with a long rubber tube to assist by siphonage. Southey's capillary trocars may be used when a very gradual removal of the fluid seems advisable. All of these methods may fail to empty the pleura, and therefore most operators prefer an aspirator. Two forms of this instrument have their advocates—Dieulafoy's syringe aspirator and Potain's bottle aspirator. The latter is more rapid and less laborious to work owing to the larger vacuum engaged, but Dieulafoy's instrument is the safer, as it can be more accurately gauged and controlled in the event of pain, bleeding, or syncope supervening.

It is assumed that the instruments have been sterilised, and that the hands of the operator and the skin of the patient have been cleansed with soap and water, rubbed with ether or alcohol, and finally moistened with carbolic lotion 1 in 40, or a mercurial lotion 1 in 1000. The patient should be recumbent or nearly so. No general anæsthetic is needed, even in children; and local anæsthesia by freezing or cocaine is also best avoided, as the former hardens the tissues, and the latter may cause a misleading syncope.

The puncture should not be made too low down, lest the abdomen be entered. Any interspace from the fifth to the eighth inclusive on the left side, and either the fifth or sixth on the right will suit, as the vacuum renders the operation independent of gravitation. The needle should be of good size,  $\frac{1}{12}$ – $\frac{1}{10}$  inch, and the trocar and cannula mount is preferable to the single needle, as the cannula can be cleared if blocked by coagula.



Guarded by the forefinger, and guided by the left forefinger pushed into the interspace, the needle should be confidently thrust through the middle of the space. A hesitating movement is apt to push the thickened pleura before the needle and fail to lodge it in the cavity. For a similar reason a hypodermic syringe, often used in the preliminary exploration, is not to be recommended, as from the shortness and fineness of its needle and feeble suction it may fail to reach and draw a sample of the fluid.

The evacuation should proceed slowly, particularly in left-sided cases in which there is marked displacement of organs; and if bleeding, pain, spasmodic cough or syncope arise it will be well to suspend or stop the operation. In the event of bleeding, it is unwise to resume; but in the three other conditions mentioned, the operator may judge by their severity and persistence whether he may proceed. In the absence of these symptoms, it is best to wholly empty the cavity, as the absorption of the residue, after removal of a part of the fluid, does not occur so frequently as to entitle one to calculate upon its occurrence.

At the close the puncture may be sealed with an antiseptic pad and strapping, or it may be painted with collodion. It is neither necessary nor desirable to bind the chest after aspiration; on the contrary, the respiratory movements should be encouraged.

Difficulties in the operation may arise from close approximation of the ribs, thickening of the pleura, a multiloculated condition, and much coagulation of the fluid. Care should be taken not to wound the heart, pericardium, or the abdominal organs. Wounding of the intercostal vessels need not be feared. Two special risks require more detailed notice, viz. syncope and acute pulmonary oedema. Syncope is not uncommon during the progress of the operation, and is to be promptly combated by diffusible stimulants, nitrites, and by hypodermic injections of strychnia and ether.

Acute oedema of the lung (*L'expectoration albumineuse*, Terrillon), with copious expectoration of serum, is believed to be due to the sudden release of the lung from pressure, with vaso-motor changes and consequent flooding of the air vesicles with serum strained off from the dilated capillaries. It is an alarming condition and fortunately rare. We are unable "to foresee, prevent, or alleviate it" (S. Paget). Oedema is more likely to follow the too rapid evacuation of a large effusion, and the onset may be immediate, or be delayed for a few minutes or an hour or two. The duration of the attack may only be a few minutes, or extend to hours or days. The patient becomes cyanosed, oppressed in breathing, with incessant cough, and may expectorate in an hour or two one to three pints of frothy serum. Fine moist râles are heard over the lung. A fatal termination is not infrequent, but many recover. Prompt treatment by alcohol, the nitrites, and strychnia must be instituted, and dry cupping and oxygen inhalations may also be administered.

*Drainage of chronic pleurisies* that demand repeated aspiration has been much debated; and although successful cases have been recorded by West, Morison, Wilson, and others, it must be admitted that this step has not yet received wide surgical approbation. The almost inevitable conversion of such pleurisies into empyemata, with persistent discharge, renders the propriety of incising and draining these cavities very doubtful. The writer's experience is wholly in favour of depending upon repeated aspiration in such cases.

**EMPHYEMA** — *Surgical Treatment of.* — It should be remembered that purulent collections in the pleura are usually loculated; in a smaller



number of instances the pus occupies the entire cavity; some are multilocular; and a few are bilateral. Of the bilateral effusions one side may contain serous fluid and the other pus.

Recently it has been sought to determine the presence of pus by the X-rays (Walsham, Kilian), but in the present state of our knowledge the aspiration of a sample of the fluid is still needed for proof.

The classification of empyemata into five forms, according to the micro-organism engaged in their production, has an important bearing upon surgical management and prognosis.

1. Pneumococcus empyema.

2. Streptococcus                    „

3. Saprogenic                        „

4. Tubercular                        „

5. Staphylococcus                 „

The last-named is often variously mixed with the other forms, and does not often appear as a separate pathological entity (Netter).

While it cannot be denied that one or more exhaustive aspirations may cure a pneumococcal empyema, especially in children, it will commonly be found necessary to provide a free opening with drainage, either by incision through an intercostal space, or the excision of a segment of a rib. Certainly in streptococcal, saprogenic, and staphylococcal empyemata no delay is warrantable, as the pus is sure to re-accumulate after aspiration, and the risk to life of the retention of these kinds of pus is greater than in the pneumococcal variety, which is the most hopeful of all. On the other hand, the writer has seen nothing but misfortune result from these open measures applied to tubercular empyema, and he strongly endorses the view that aspiration, repeated only so often as the dyspnoea demands, instead of permanent drainage, is the best course to adopt in these very unpromising cases.

*Anæsthesia* in these operations is always an anxious question, particularly so in patients with much dyspnoea and displacement of organs, and in those with recently-established broncho-pleural fistula. The anæsthesia should never be profound; and indeed, in both its general and local forms it may occasionally be dispensed with. Chloroform or methylene is more suitable than ether, as the latter embarrasses by the coughing and salivation which it induces. A preliminary aspiration to reduce the excessive quantity of pus is often a wise precaution.

The patient should be recumbent, and rolled over on the sound side as little as possible. The opening should be made in the eighth interspace just in front of the scapular angle, or the ninth rib may be resected in the same situation. Efficient drainage both in the erect and lying postures is thus provided, and it will seldom be found necessary to make two thoracic openings as was formerly the practice recommended. In small loculated empyemata the opening must of course be made where the dulness indicates, and in all cases the presence of pus at the selected spot must be verified by an exploratory puncture at the time of operation.

Excision of a rib is superior to mere incision of an interspace, for it supplies an opening which does not become valvular, and which admits a finger for examination of the cavity and lung and the evacuation of large fibrinous coagula. Moreover, the advantage of easy dressing in children and nervous adults is obvious. The risks of the bony section favouring pyæmia, etc., are fanciful.

*The Operation.*—In incising an interspace the soft parts are to be steadied upon the lower rib bounding the interspace with the left forefinger and thumb,



and an incision two and a half to three inches long is made down upon its upper border, thus avoiding the intercostal vessels and nerve. When all bleeding points have been secured, the pleura is opened by a small incision, which is widened by the insertion of a dressing forceps. The finger is then introduced to prevent a too rapid escape of the pus, and to assist in dislodging coagula.

Excision of the rib involves a three-inch incision boldly carried down to the middle of its outer surface, dividing the periosteum with the other soft tissues. The periosteum is raised towards the rib borders with an elevator for two inches, and the intercostal muscles are detached for a like distance, with due regard to the vessels and nerve, which are practically never seen. The periosteum is then cautiously separated from the deep surface by a lateral movement of the elevator, and a segment of bone, about an inch and a half in length, is clipped out, the elevator being used as a lever to facilitate this step. After securing the bleeding points, the pleura is opened as above described, and the finger is introduced to ascertain the degree of lung recession and to rake out fibrinous coagula.

At this stage the propriety of washing out the cavity with normal saline solution at 100° Fahr., or boracic acid solution, or other non-poisonous antiseptic, may be considered. The frequent sudden fatalities resulting from such lavage, and the clinical observation that even the most putrid discharges become sweet in a few days by thorough drainage, justify a condemnation of this practice as a routine measure. If ever employed, the irrigating funnel and tube should be used instead of a syringe, and the lotion allowed to flow into the cavity in the gentlest stream, a tube of larger calibre having been inserted to serve as an outflow, in order that no compression of the lung by the irrigating fluid may take place.

No sutures are required for the wound. The permanent drain tube is placed in position and lightly packed around with iodoform or cyanide gauze, a copious absorbent pad of wood-wool tissue is placed over the gauze, and a binder or many-tailed bandage is applied over all.

Drainage tubes of metal, vulcanite, glass, or rubber with a metallic core have been advocated; but a simple rubber tube, one-third or one-half inch in diameter, and furnished with a sheet-rubber or gutta-percha shield to prevent it slipping into the cavity, answers all purposes. Such a tube should have no lateral perforations, save one near the free end, and it should project about one inch into the pleural cavity. There is no advantage in long tubes; but when much flocculent material mingles with the pus, two tubes of slightly different lengths, arranged parallel, may occasionally obviate blocking.

*Bilateral empyema* should be approached with caution, for, if extensive pleural adhesions have not formed, it will not be safe to open both pleuræ at the same time. One side should be aspirated and the other incised. After an interval of a week or more the aspirated side may be freely opened also.

*Treatment after Operation.*—The dressings may be changed twice daily for a few days, and later once a day will suffice. The tube, if patent, need not be disturbed for a week. The time at which it can be permanently dispensed with varies with the amount and character of the discharge. One week in children and three weeks in adults may be regarded as minimal. A safer guide is the amount of the discharge; when this declines to half an ounce or less, and becomes almost serous in kind, the tube may be discarded. Godlee's plan of passing a railroad catheter every third day, for a short time thereafter, to prevent accumulation, may be borne in mind. The patient may sit up, if strong enough, in a week or ten days after operation; and may get out-of-doors in three weeks, provided there is no fever.

*Causes of Death in Empyema.*—Death may result from increasing embarrassment of breathing in the unrelieved cases. In connection with anæsthesia, operations, or lavage, the catastrophic deaths have already been alluded to. In chronic cases with discharge, septic poisoning, pyæmia with visceral abscesses, hectic fever, amyloid disease, and recurrent hæmorrhages from the giving way of pleural adhesions, may all account for fatal terminations.

*Prognosis* is very favourable in the acute empyema of children, in whom the pneumonococcal form is so common. In adults this form is also very hopeful, if treated early. The streptococcal, staphylococcal, and saprogenic forms are more dangerous; but often do well, if uncomplicated by visceral



disease or general septic states. The tubercular variety is practically incurable, in so far as cessation of discharge is concerned, but the fatal issue may be delayed for months or years.

As to the relative frequency of these forms, Netter's analysis of 110 cases showed—32 of the pneumonococcal; 51 of the streptococcal, often combined with the staphylococcal; 15 of the saprogenic; and 12 of the tubercular.

**THORACOPLASTY.**—*Estländer's Operation—Thorax Resection.*—Five factors are concerned in the obliteration of the empyema cavity—viz. the expansion of the lung, adhesions between the pleural layers, contraction of the granulation tissue tending to draw the lung and chest wall together, the elevation of the diaphragm, and the falling in of the ribs from atmospheric pressure. To favour the last of these, in intractable cases, Estländer devised the operation of resecting portions of those ribs that bound the cavity. The number and length of the segments requiring removal are determined by flexible probes passed through the sinus, and during the operation by the finger itself.

One long incision over the central rib will admit of three ribs being excised; but, where a larger number of ribs is condemned, several incisions may be needed, or, better, a large flap of the thoracic skin and muscles may be turned up. The ribs are subperiosteally excised for the required distance, and the flap or wound edges sutured, leaving ample drainage openings for tubes or gauze tampons. Schede suggested an actual "thorax resection." Turning up a large musculo-cutaneous flap, with its base upwards, and extending from the fourth rib in front down to the tenth rib in the axillary line, and up along the vertebral border of the scapula, Schede resects the ribs from their tubercles to the costo-chondral junctions. Though the ribs are subperiosteally excised to save hæmorrhage, the periosteum is subsequently excised together with the intercostal muscles and the thickened parietal pleura, forming the whole outer wall of the cavity. He then replaces the scapula and flap, which have meanwhile been strongly retracted. The operation is a severe one, attended by much shock and hæmorrhage, and the results are often disappointing. Schede's tables of his operations, showing ten cases with eight cures and two deaths, are very encouraging, but the experience in this country, while not unfavourable to its adoption in selected cases, is not such as to lead to so sanguine an estimate of its general applicability and value. It must be conceded that no other operations remedy the spinal curvature or restore the figure in so marked a manner as these extensive resections, but, on the other hand, many of these resections are only partial obliterations of the cavity, the apical portion remaining a suppurating space requiring permanent drainage and resisting all attempts at closure by skin grafting and other plastic measures. The comparative immobility of the two upper ribs is responsible for the failure. The first rib has been resected, but it is considered unwise to tamper with it; and the sinkage of the second on resection is very trifling and the result disappointing. As a guide to the selection of cases for these extensive resections it may be stated, that marked spinal deformity, copious exhaustive discharge, or recurrent hæmorrhage form indications; but the best chance of success is afforded by single cavities of moderate size, bounded by the lower and more moveable ribs, in subjects under middle age who are free from tubercular taint.

It must not be forgotten that chronic empyema patients occasionally live for fifteen to twenty years, and may be able to follow their occupations notwithstanding a daily discharge of pus.



Certain difficulties that occasionally complicate the management of empyema remain for consideration.

1. Natural openings in unfavourable situations may have formed, and the advantage of making an artificial drain opening in the situation recommended may in such cases be considered. With our present knowledge, the natural pointing of the empyema ought theoretically never to be seen. Marshall long ago pointed out its frequent occurrence in the second and fifth interspaces in front.

2. Broncho-pleural fistula may exist, and if after a week or two it is evident, from the continuance of septic fever, sweating, and cough, that the cavity retains much pus, a suitable empyema operation has much to recommend it, but caution about administering a general anæsthetic will not be misplaced.

3. Abscess of the brain, "pulmonal cerebral abscess," is said to occur in about 8 per cent of suppurations in the pleura and lung. It is probably pyæmic, and does not lend itself favourably to trephining, for it may be multiple and difficult to localise. If the signs are clear, it would be right to trephine and drain it.

4. A differential diagnosis must be made from pyo-pericardium, subphrenic abscess, and hydatids. Reference to the special articles upon the pericardium and subphrenic abscess may be made for their respective signs, but a brief notice of pleural hydatids is introduced below.

HYDATIDS OF THE PLEURA may be primary, or they may extend to the pleura from the lungs or liver. The favourite seat is the lower part of the right pleura. They may attain a large size or may burst into a bronchus, the patient coughing up characteristic elements, which, if mingled with bile, point to a hepatic origin. If a hydatid suppurate, the signs and treatment resemble those of empyema, and the prognosis, when similarly incised and drained, is good. Before suppuration signs of a localised collection of fluid having a convex upper outline may be detected, and these signs, associated with considerable pain, but no fever, may excite suspicion of hydatids. Probably aspiration will have been carried out before a diagnosis is made. In some cases, however, transient attacks of slight fever as the hydatid grows may be met with. As regards treatment, it is to be remembered, that aspiration in hydatids has special risks of swamping the lung and thus suffocating the patient. If, therefore, a strong probability exist that the disease is hydatids, free incision, with removal of the cyst, should be preferred. In chronic cases wherein the cyst cannot be extirpated prolonged drainage must be employed.

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**Pleurodynia.**—This term is applied to the pain of a so-called rheumatic nature that affects the muscles, and fibro-tendinous structures of the chest wall. It is commonly spoken of as muscular rheumatism. Its ætiology will be discussed later (see "RHEUMATISM"). The clinical features are fairly distinct, and consist of a sharp, sudden, and often lancinating pain in the intercostal space. In some cases the pain is very acute, and cough is a frequent accompaniment. On physical examination little may be found, but in well-marked cases there is an area of acute



tenderness in the intercostal space or in the pectoral or serratus muscles. Pain is aggravated by the respiratory movement. The differential diagnosis has to be made from intercostal neuralgia and cardiac, pulmonary, or pleural disease. The former is characterised by the presence of localised painful points corresponding to the entrance of sensory nerves; the latter must be determined by looking for the ordinary physical signs of cardiac or pulmonary disease. The local treatment consists in the application of a collodion dressing, or strip of plaster. Some cases are much benefited by counter-irritation. Only in specially severe cases will it be necessary to give an opiate for the relief of the pain. The disease is a constitutional one, and the general treatment will be described under "RHEUMATISM" (*q.v.*).

## Pneumonia.

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### Bacteriological Section

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INTRODUCTORY.—The term pneumonia is properly applied to any inflammatory process affecting the lung tissue, just as pleurisy is employed to designate any inflammation of the pleural membranes, or as hepatitis and nephritis are applied to any inflammation of the liver and kidneys respectively. Moreover, as there may be acute or chronic parenchymatous or interstitial forms of hepatitis and of nephritis, so there may be corresponding forms of pulmonary inflammation or pneumonia. In the more important forms of chronic pneumonia the interstitial changes especially predominate, while in the acute pneumonias the more characteristic lesions are as a rule due to the implication of the pulmonary parenchyma. Any acute inflammation of the lung, therefore, is an acute pneumonia, but it is usual to recognise several varieties.

Sometimes the classification is based on anatomical differences in the resulting lesions. When the inflammatory process is specially concentrated around the smaller bronchi, and follows their lobular distribution, the term broncho-pneumonia or lobular pneumonia is employed. The term catarrhal pneumonia is often applied to the same process, owing to the fact that the exudation into the pulmonary alveoli largely consists of catarrhal cells. More rarely the acute inflammatory changes select the connective tissue framework of the lung, giving rise to an acute interstitial form of pneumonia, such as is met in some forms of influenzal pneumonia and in pleuropneumonia of cattle. A third type is recognised under the name of acute lobar, or fibrinous, or croupous pneumonia when the inflammatory



process involves large continuous tracts of lung tissue, such as an entire lobe, or the greater part of a lobe, and when the exudation into the pulmonary alveoli is at first and for the most part composed of fibrin. As a matter of fact, however, there are commonly met forms of acute pneumonia which do not conform to any one of these anatomical types, but present very variable and mixed lesions. Even among those which are rightly classified as lobar forms of acute pneumonia there may appear very striking differences as regards the precise anatomical lesion.

Other forms of acute pneumonia are differentiated with special reference to the causal agent associated with them. Such specific forms as the tubercular inflammations of the lungs, or such forms as the suppurative pneumonia of pyæmia, present, as a rule, definite types with characteristic anatomical lesions. But when we try to apply this ætiological differentia to other forms of pneumonia we encounter the difficulty that the same cause may give rise to anatomically different pulmonary lesions. Thus a group of acute pneumonias due to the pneumococcus would include both bronchopneumonic and lobar types. A similar difficulty is presented by the forms of pneumonia that appear in influenza, plague, and many other conditions of microbic infection. It is, therefore, better, in place of attempting an exact definition or a precise outline of the scope of this article, merely to state that in discussing the pathology of acute pneumonia it is understood that the term is limited to certain forms of acute pulmonary inflammation, all microbic in origin, and probably all due to or associated with Fraenkel's pneumococcus, but that all forms of the typical lobar type are necessarily included.

**HISTORICAL.**—Long before any bacterium had been associated with acute lobar pneumonia as the causal agent it had been suspected that this condition was infective and analogous in every way to the acute specific fevers. Its occurrence in epidemic form, and some instances apparently of direct infection, together with the definite course of the fever, all tended to support this view. But it was not until 1883 that the first important contribution to the bacteriology of acute pneumonia was made.

In that year Friedländer published a paper which attracted much attention, and in which he claimed to have obtained in pure cultures the causal microbe of pneumonia. This he described as a coccus, usually occurring in pairs, and surrounded by a definite capsule. They were found in the lung tissue, bronchi, etc., in cases of acute pneumonia. They could be isolated and grown on peptone gelatine at the ordinary room temperature. From this fact alone it is obvious that the coccus described and isolated by Friedländer is not the same as that which is now accepted as the most important ætiological factor in acute pneumonia and is associated with the name of Fraenkel. In fact the organism which Friedländer at first described as a coccus has now come to be regarded as a short bacillus, and is commonly known as Friedländer's pneumobacillus. Inoculation of animals with Friedländer's organism, however, in many instances caused death from septicæmia with inflammation of the serous membranes, thus demonstrating the pathogenic capacity of the organism in certain conditions. One of the most important results of Friedländer's work was the stimulus it afforded to the study of the bacteriology of pneumonia.

In the same year (1883) Talamon described a coccus which he obtained from the sputum and pulmonary exudation in acute pneumonia, and which from its shape he termed "lanceolatus." This organism he was able to cultivate in bouillon, but usually in an impure condition, and inoculation of rabbits produced a septicæmia often associated with a fibrinous pericarditis and pleurisy. There can be little doubt that this organism is the same as the pneumococcus later described and isolated by Fraenkel.

As early as 1880 certain observations were made in America by Sternberg and in France by Pasteur on the presence in the saliva in health and in disease of micrococci, and on the fact that inoculation of rabbits with saliva gave rise to



a "sputum septicæmia," in which the blood teemed with micrococci. The significance of these observations was not at first appreciated.

After a preliminary communication in 1884 Fraenkel published in 1886 a very full and accurate description of the characters of the pneumococcus—noted its lanceolate outline, its occurrence in pairs, and its capsulated appearance. He succeeded in isolating pure cultures, and found that both in its cultural and pathogenic characters the microbe differed from that previously described by Friedländer. In particular, Fraenkel's organism did not grow on gelatine at the ordinary room temperature, and it was highly pathogenic to rabbits, in both these respects contrasting with Friedländer's organism. Another important advance made by Fraenkel was the identification of the pneumococcus with the "coccus of sputum septicæmia" previously described in healthy saliva by Pasteur and Sternberg.

In the same year (1886) Weichselbaum independently published the results of a still more extensive research on the bacteriology of acute pneumonia, including ninety-four cases of the typical lobar form and thirty-five other cases of acute pulmonary inflammation. From these he isolated four organisms: (1) The "diplococcus pneumoniae," which he described as an oval or lanceolate coccus, corresponding in morphological, cultural, and pathogenic characters with Fraenkel's pneumococcus; (2) the "streptococcus pneumoniae," which, on the whole, presented similar characters, but was rounder and formed longer chains; (3) the "bacillus pneumoniae," which he regarded as identical with Friedländer's pneumobacillus; (4) the staphylococcus pyogenes aureus. Of these organisms the "diplococcus pneumoniae" was present in the great majority of cases, the "streptococcus" was much less common, and the "bacillus pneumoniae" rarer still. He was led to the conclusion that while the great majority of cases of acute pneumonia are due to the "diplococcus pneumoniae" (Fraenkel's pneumococcus), some are caused mainly by a streptococcus, and a few by the "bacillus pneumoniae" (Friedländer's pneumobacillus). How far the streptococcus pneumoniae of Weichselbaum corresponds with the ordinary streptococcus pyogenes, and how far with Fraenkel's pneumococcus, is left somewhat uncertain; and what is of more importance, the reader is left uncertain as to the data on which his conclusions are based, in particular, whether they have reference to acute lobar pneumonia in its limited acceptance or acute pneumonia in its widest sense.

Since these earlier researches were published the attention of many observers has been devoted to the elucidation of problems involved in the relation of microorganisms, especially the pneumococcus, to acute pneumonia. Among the later contributors may be noted Gamaleia, who investigated in particular the pathogenic properties of the pneumococcus in experimental inoculation; Netter, who examined a great number of cases with the object of determining the distribution of the pneumococcus and its relation to various morbid states; Eyre and Washbourn, who perfected the culture media, and the latter of whom was successful in establishing a condition of artificial immunity.

THE PNEUMOCOCCUS (Fraenkel).—It is obvious from these researches that whether this organism is admitted to be the essential causal agent in the pathogenesis of acute pneumonia, or whether it acts thus only in the majority of cases, it is of all the microbes concerned by far the most important. A brief outline of some of its morphological, biological, and pathogenic characters is a necessary preliminary to any discussion regarding its rôle in the production of disease in man, and the whole question of its relationship to disease introduces one of the most interesting problems which bacteriology has ever attempted to solve.

In form the pneumococcus appears as minute, oval, or lanceolate bodies, usually in pairs and often in chains, the free extremities of each pair being pointed. In animal tissues and fluids each pair of cocci appears surrounded by a capsule, due to a peculiar swelling of the cell envelope, and this capsule may be stained by special methods. In any circumstances, but especially in artificial culture, the cocci show a marked tendency to degenerate and undergo involution; but even when this degeneration has advanced to such an extent that the bodies of the cocci have largely or entirely disappeared, the capsules often persist and may be identified by



suitable methods of staining. The significance of this lies in the fact that at such a stage cultures would probably remain sterile, and inoculations would probably have negative results, so that the recognition of the capsules might be the only means of determining the presence of pneumococci. In contrast with the pneumobacillus of Friedländer it should be noted that the pneumococcus retains the stain in Gram's method.

In cultivation the organism is somewhat fastidious, and practically requires to be incubated at or about the body temperature. On blood serum, blood agar, or ordinary agar it occurs as minute translucent colonies which tend to remain separate. On all ordinary media cultures rapidly degenerate, rapidly die, and subcultures soon lose their virulence. Eyre and Washbourn, however, by the most careful attention to certain conditions of cultivation, were enabled to maintain both the vitality and the virulence unimpaired for several months.

Both within and without the animal body the pneumococcus is quite exceptional in its brief duration of life and in the rapidity with which its virulence is lost. In artificial culture this is very striking, and presents a considerable difficulty in the way of experimental work. Sometimes the virulence may be intensified or restored by passage through a series of rabbits. In susceptible animals the organism may live longer in a virulent condition than in cultures, but even then it is apt to die sooner than most pathogenic microbes. In old empyemas, in the later stages of pneumonia, etc., a large number of devitalised and degenerated pneumococci are often found, but occasional instances are met where the virulence has been maintained for months.

Fraenkel and Weichselbaum had stated, and their views have been confirmed by subsequent observers, that pneumococci suffer a progressive diminution in virulence as the course of a pneumonia advances. Netter, indeed, goes so far as to say that in the day of the crisis the virulence of the sputum is often suddenly lost, and remains in abeyance for two or three weeks. Apropos of this question Welch of Baltimore says that attenuation of virulence within the human body probably does occur, inasmuch as pneumococci derived from inflammatory exudates may be devoid of any pathogenic effect in animals. From the late stages of pneumonia which had gone on to recovery, and at the time of the crisis, he had obtained pneumococci which were as virulent and sometimes more virulent than those procured from earlier stages of the same case. He points out that in experiments of this nature the number and character of living pneumococci inoculated must be considered. It is known that the pulmonary exudate contains many dead cocci, and that the living cocci vary greatly among themselves in degree of virulence. Moreover, when animals are inoculated with material obtained directly from the human body, there may be introduced along with the pneumococci toxic or immunising substances which influence the course of the injection. It may indeed be shown that such material, though proved to contain very virulent pneumococci, may not produce any pathogenic effect.

*Pathogenic Effects of Experimental Inoculation.*—The results of experimental inoculation of animals with the pneumococcus show extreme degrees of variation. The actual number of cocci injected, the presence or absence of other bacteria or toxins, and the site of inoculation, very materially condition the result. But the two factors on which this variability chiefly depends are (1) the virulence of the microbe and (2) the susceptibility of the animal.

(1) Both in natural conditions and in artificial cultures pneumococci may have lost their virulence to such an extent as to be quite innocuous. On the other hand, by such methods as passage through a series of susceptible animals, the virulence may be so intensified that a few cocci injected into a rabbit will produce a fatal septicæmia in less than twenty-four hours. Between these extremes pneumococci of all degrees of virulence may be met, so that the possibilities of variation in the lesions produced by this cause alone are obviously considerable. Of course if the resistance of the animal is increased the effects are similar to those due to attenuation of the virus. Thus a rabbit which has received a subcutaneous inoculation



of virulent pneumococci usually dies in about forty-eight hours of an acute septicæmia. In such cases the local inflammatory reaction at the site of inoculation is always slight, but the blood is found to be swarming with enormous numbers of capsulated pneumococci. If, however, a rabbit which has been partially immunised receives a similar injection, or if a somewhat less virulent organism is employed, the animal may live for a week before it succumbs to the infection. Here the local reaction is more intense, so that at the site of inoculation the tissues show an extensive area of inflammatory œdema, hæmorrhage, and frequently also necrosis. The fluids at the local site are crowded with pneumococci, but the blood may contain few or none, or may show the results of a septicæmia which has immediately preceded death. A third and entirely different result may be obtained if the rabbit be still more highly immunised, or if the pneumococci be still more attenuated. In such conditions a local focus of suppuration forms at the site of inoculation, and the animal usually recovers. Hence in the same species of animal, the rabbit, by suitable modifications of its powers of resistance or of the virulence of the pneumococci, subcutaneous inoculations may result in such divergent lesions as (*a*) an acute septicæmia with a negligible local reaction, (*b*) an intense local cellulitis, œdema, and necrosis with little tendency to septicæmia, and (*c*) a localised abscess of varying size.

(2) When the susceptibilities of different orders of animals are also considered, the interpretation of the pathogenic effects of the pneumococcus becomes still more complicated. Among the most susceptible are mice and rabbits, and these animals when inoculated beneath the skin, in the peritoneal sac, or in the lung, with virulent pneumococci, almost invariably die of an acute septicæmia with little or no local reaction. Less susceptible are guinea-pigs, sheep, and dogs, in which animals subcutaneous inoculation results in an extensive local cellulitis and œdema which does not invariably lead to a fatal result. If, however, in a partially-immunised rabbit, or in the more immune animals, such as the sheep or the dog, the injection of pneumococci be not subcutaneous, but intra-pulmonary, then again an intense local inflammation ensues at the site of inoculation, which in this case is the lung, and an acute fibrinous pneumonia and pleurisy results. "When a dose of pneumococci sufficient to kill a rabbit is injected subcutaneously in the human subject, it gives rise to a local inflammatory swelling with redness and slight rise of temperature, all of which pass off in a few days. It is, therefore, justifiable to suppose that man occupies an intermediate place in the scale of susceptibility, probably between the dog and the sheep, and that when the pneumococcus gains an entrance to his lungs the local reaction in the form of pneumonia occurs" (Muir and Ritchie).

*Intoxication and Immunity.*—It is a noteworthy fact that few cases of pneumonia succumb to asphyxia, so that the extent of lung involved is not in most instances the direct cause of death. What is probably of more effect in determining a fatal issue is the degree of nervous prostration, and especially of cardiac failure. After death, in addition to the pulmonary lesion, there is invariably evidence in the condition of the heart, spleen, and kidneys, and other viscera, that the patient has suffered from the effects of an acute general intoxication. During life, moreover, many of the more urgent symptoms are referable to such a condition. It rarely happens that any general invasion of the blood by pneumococci occurs except just before death. It is, therefore, more than probable that pneumococci, which invade the lung and there produce an acute local inflammation, also elaborate



some diffusible toxic substance which, absorbed into the blood-stream, gives rise to the general toxæmia of an acute pneumonia. The nature of these toxic bodies is not definitely known, but without going into details, one may say that all experimental work on the subject supports this view of their existence and highly poisonous nature.

Artificial immunisation of susceptible animals has also been effected by various observers, including G. and F. Klemperer and Washbourn. Moreover, it was found that the blood serum of animals artificially immunised was itself protective, in other words, that a true antitoxin was produced. It is supposed that the crisis so typical of the recovery from an acute lobar pneumonia occurs only when the antitoxin has been developed in such abundance as completely to neutralise the toxin. In this relation two facts are of significance as supporting the view that the pneumococcus is the essential cause of acute lobar pneumonia. (1) The serum of patients who have recovered from such an attack is to some extent protective against the results of artificial inoculation with the pneumococcus. (2) The serum of an animal artificially immunised by injection of cultures of pneumococci has a beneficial action in cases of acute lobar pneumonia.

*Distribution of the Pneumococcus in Health and in Disease.*—Reference has already been made to the fact that the saliva of healthy persons injected into a rabbit may produce a fatal septicæmia, due to the presence of pneumococci. The existence of virulent pneumococci in the mouth in health has obviously a most important bearing in the pathogenesis of acute pneumonia.

In the production of diseased conditions it is not easy to exaggerate the rôle of the pneumococcus, for the multiplicity and frequency of the lesions that may be ascribed to its action are indeed remarkable. Pulmonary inflammations are undoubtedly the most common effect, and, in the opinion of the writer, pneumococci may be found in every case of acute lobar pneumonia, in most cases of broncho-pneumonia, and in most of those more or less mixed forms of pneumonia which are complicated by the associated action of other organisms, such as influenza, plague, typhoid, diphtheria, etc. In cases of typical lobar pneumonia they are practically the only organisms present in the affected lung, and are found in greatest abundance in the fibrinous exudation into the pulmonary alveoli and in the pleural surface. They are most readily detected in and isolated from the more recent parts of the pulmonary lesion, as in the older parts they are usually much degenerated. In the purulent softening and other suppurative changes that appear in the lung in many fatal cases of pneumonia, they may be the only organisms present, or they may be associated with other pyogenic bacteria, especially streptococci.

Next to the lungs the cerebro-spinal membranes are most frequently attacked, and this may occur not only as a sequel to or a concomitant of pneumonia, but as an apparently independent condition. The resulting meningitis is sero-purulent and usually very widespread, involving both cerebral and spinal membranes. The serous cavities of the thorax and abdomen are frequently the seat of pneumococcal infection by direct extension from an affected lung, but they too may show an apparently primary and independent inflammation. Empyema of pneumococcal origin is comparatively common, and, in the experience of the writer, most cases of fibrinous pericarditis with sero-purulent effusion are due to pneumococci, whether an antecedent pneumonia can be traced or not. Primary peritonitis of pneumococcal origin is uncommon, but a recent peritonitis in fatal cases of pneumonia is by no means rare. In children one of the



most common effects is an otitis media, and in children, too, broncho-pneumonic lesions due to pneumococci are relatively more frequent than in adults.

In the more virulent forms of pneumonia the lesions produced by the pneumococcus tend to become multiple, and the condition more nearly approaches the septicæmic type of infection, as seen most typically in mice and rabbits. In such cases not only may lungs and pleuræ be affected along with the pericardium and the mediastinal tissues, but more remote lesions are frequently associated, *e.g.* meningitis, endocarditis, arthritis, etc., and in some instances pneumococci are found in considerable numbers in the circulating blood.

*Summary and Conclusions.*—One result of these laborious researches is to show that the pneumococcus produces in man a local lesion, usually pulmonary, with general toxic effects. Sometimes the local lesion takes the form of a meningitis, an otitis, an empyema, or a pericarditis, as an apparently independent condition. This is really the same disease as acute lobar pneumonia with an accidental difference in the site of inoculation and consequent local inflammatory reaction, and many of the symptoms are similar from similar toxic effects. Hence it not infrequently happens that a pneumococcal pericarditis or empyema is mistaken for an acute pneumonia. Occasionally, however, the pneumococcus acquires increased virulence, and produces a condition more allied to the septicæmia of mice and rabbits.

It is difficult to avoid the conclusion that the pneumococcus is the essential cause of acute lobar pneumonia, as well as of the many other inflammatory conditions with which it is associated. But the versatility of this remarkable organism renders the proof much more complex than in the case of pathogenic bacteria. At first sight, too, Koch's elementary postulates for establishing a causal relationship appear to be all evaded by the pneumococcus. For it cannot be said to be present in all cases of acute pneumonia, and in a few instances even of typical lobar forms it has been sought in vain. Moreover, it is present in the mouth and upper respiratory tract in health as well as in disease. Again, although it has been isolated in pure cultures, inoculation of animals does not reproduce the identical lesions found in man. And, finally, there is no consistent lesion even in the human subject, and not even in the lung.

The allegation that the pneumococcus is occasionally absent even in typical cases of acute lobar pneumonia is one which is open to doubt. For, if the statement is founded on a microscopic examination, it might be that none of the cocci would be visible, having undergone degeneration until only capsules were left. Again, if based on the negative results of cultures, the fallacy might be due to the devitalised condition of the cocci. Even if reliance is placed on the negative results of experimental inoculation of lung tissue, it must be remembered that, after a time, the virulence of pneumococci in the animal body is often quite lost, or that antagonistic substances, of the nature of antitoxins, may have been injected along with the cocci, and so neutralised their effect.

The fact that pneumococci are usually present in the saliva of healthy persons has proved a stumblingblock to many writers, but this is precisely the fact required to explain the incidence of pneumonia from exposure to cold, and as a sequel to debilitating diseases and exhausting fevers. Apparently the cocci in the buccal and nasal secretions are not sufficiently virulent to produce any pathogenic effects in health. But, when the resistance is lowered by some depressing influence, the organisms are enabled to



infect the tissues and develop inflammatory conditions. From their anatomical positions the lungs would be most easily infected, but the readiness with which an otitis or a meningitis might be developed from the nares is also obvious.

The contention that inoculation of pure cultures does not sufficiently reproduce the disease in animals is quite untenable when the following points are considered:—(1) The effects of inoculating pneumococci of varying virulence in animals of varying susceptibility; (2) the fact that, in the human subject, the lesions admittedly due to pneumococci are almost as varied as those produced in animals by artificial means; (3) the analogy with such organisms as the bacillus anthracis, which causes in the susceptible ox or guinea-pig an acute septicæmia, and in the more immune human subject an intense local reaction.

The fact that even in the lung in man there is no constant lesion—the pneumococcus being associated sometimes with an acute lobar pneumonia, sometimes with a broncho-pneumonia—is more difficult to explain. It has been suggested that variations in virulence and in susceptibility might modify the result, and probably also the precise mode of entrance to the lung would condition the anatomical form of the lesion.

**OTHER BACTERIA ASSOCIATED WITH ACUTE PNEUMONIA.**—Although, as has been shown, there is good reason to believe that the pneumococcus is the essential cause of all typical forms of acute lobar pneumonia, there are many other conditions in which acute pneumonia may arise. Some of these forms are broncho-pneumonic in type, others are of a mixed character, and of both of these it may be said, either (*a*) that they are due to the pneumococcus alone — broncho - pneumonic forms of pneumococcal infection especially predominating in children, or (*b*) that they are due to other bacteria associated with the pneumococcus, or (*c*) that they are due to other bacteria alone. Among the various bacteria which may be the cause of acute pneumonia (other than typical lobar forms), and which may act either in conjunction with, or independently of the pneumococcus, are Friedländer's pneumobacillus and those associated with the following diseases: influenza, plague, diphtheria, enteric fever, septicæmia, and pyæmia.

Friedländer's pneumobacillus is of special interest as being one of the original claimants for supremacy in the production of acute lobar pneumonia. In 1886, when the fundamental papers of Fraenkel and of Weichselbaum appeared, the former concluded that the pneumococcus was the sole cause of true lobar pneumonia, while the latter held that about 5 per cent was due to the pneumobacillus alone. Each view still has its own supporters, but most recent work is in the direction of showing that the pneumobacillus, though frequently associated with the pneumococcus, is not the essential factor in typical lobar forms. A short bacillus, usually arranged in pairs, and in body fluids and tissues usually encapsulated, it differs from the pneumococcus in not retaining the stain by Gram's method, and in growing more readily in culture, forming the "nail" growth in gelatine. The fact that it more readily adapts itself to a saprophytic existence offers a probable explanation of the cases in which this organism alone has been cultivated from acute lobar pneumonia, for at the time when the lung tissue or secretion is examined, the pneumococcus may be so devitalised as not to grow in artificial culture, and not to infect animals on inoculation. It must be admitted, however, that the pneumobacillus is distinctly pathogenic to certain animals; rabbits appear to be immune, but guinea-pigs and mice may die of an acute septicæmia, so that the pneumobacillus may play an important part in human disease, especial acute pulmonary inflammations, though not the essential cause of lobar pneumonia.

In the acute pneumonia consecutive to influenza, the type of the lesion is mainly broncho-pneumonic, with acute interstitial changes. In the lungs and sputum the influenza bacillus has been found alone, but more frequently it is found associated with the pneumococcus. In the intense hæmorrhagic forms of



broncho-pneumonia that may occur in plague, it often happens that plague bacilli alone are found in the lung and sputum, but in many cases pneumococci, too, are present. In the broncho-pneumonia secondary to diphtheria, pneumococci are frequently present, associated with either diphtheria bacilli or streptococci, or both. In the course of enteric fever a mixed form of pneumonia may occur, in which typhoid bacilli may be present alone or along with pneumococci. In septic and suppurative pneumonias pyogenic bacteria are sometimes the only varieties found, but more frequently there is superadded an infection by the pneumococcus.

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### Clinical Section

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DEFINITION.—By the term "pneumonia" is to be understood, for the purposes of this article, a specific general infection of bacterial origin, analogous to the other infective or so-called "zymotic" diseases. This infection either begins in the air vesicles of the lungs, a primary growth of the infecting micro-organism taking place in the pulmonary parenchyma, as in a congenial soil, or else it shows a special affinity for that anatomical structure, in which it and its toxins find a secondary focus and become localised, so leading to topical, inflammatory, and sometimes organic degenerative changes. The local inflammation is signalised by increased blood-supply (or fluxion), followed in turn by hyperæmia of the affected lung substance; an exudation into the air vesicles, which become choked with it; secondary degenerative changes in that exudate; and, finally, its absorption and expulsion.

The clinical phenomena attending such an infection are, first, general; secondly, local. The general symptoms are those of a continued fever setting in after an incubation period of variable though short duration—chills, headache, rheumatoid and neuralgic pains, loss of appetite, constipation, elevation of temperature, prostration. The local subjective signs, or symptoms, are pain in the chest, hurried, difficult breathing, cough, and finally expectoration of a peculiar kind. The local objective signs, or physical signs, are such as to suggest the plugging of greater or smaller tracts of pulmonary tissue with inflammatory products, the signs changing with each progressive stage in the disease, as will be hereafter described. Evidences are usually forthcoming that neighbouring parts—the pleura on the one hand, the bronchial mucous membrane on the other—share in the mischief wrought by the disease.



Pneumonia, or pneumonic fever, as it should be more rightly called, must be regarded as a multiple infection. That is to say, it arises from one or other of many infections, as will be afterwards shown. To the classical type of acute croupous pneumonia, or lobar pneumonia, however, two micro-organisms especially seem to stand in a direct causal relation. These are—(1) The *Diplococcus pneumoniae* of Fränkel, so called by Weichselbaum, now called the “pneumococcus”; and (2) the *pneumococcus* of Friedländer, or the *Bacillus pneumoniae* of Flügge, by which latter name it is now usually known. As to the communicability of pneumonia no doubt can any longer be entertained. The spray blown from the mouth with each cough and the dried expectoration are the most probable, as they are the most obvious, channels of infection.

Usually affecting a lobe or lobes of either lung, acute pneumonia in children and in the aged is often disseminated, assuming a lobular rather than a lobar character in such patients. Like typhus fever, pneumonia is a cyclic and self-limited disease, in which the constitutional fever runs its course in five, six, or seven days, when (in uncomplicated cases) it terminates by a crisis.

HISTORICAL.—Pneumonia, taken in the widest acceptance of the term, may be described as a malady of all the centuries, of all climates, and of all ages of life. Its epidemic prevalence, according to August Hirsch, is recorded in the sixteenth-century chronicles of pestilence in Italy, Germany, the Netherlands, Switzerland, and France. One of the earliest allusions to epidemic malignant pneumonia is met with in Savonarola's *Practica*, published at Venice in 1497, where it is stated—“In civitate nostrâ Paduæ et Trevisii et Venetiæ 1440 de mense Martii antea et post apparuit *pleuresis mala*, quæ erat contagiosa, ex quâ multi et plurimi moriebantur.”

In the second book of his treatise, “Περὶ νόσων,” Hippocrates gives a faithful account of pneumonia under the name “Ἡ Περιπνευμονίη.” The later form of the word was “περιπνευμονία,” used by Celsus and Galen. Celsus (Lib. iv. cap. iii.) says—“Vehemens et acutus morbus quem περιπνευμονικόν Græci vocant.” Plutarch uses the shortened form πνευμονία, the Attic being properly πλευμονία, Hippocrates using the equivalent ἡ πλευμονίς.

Sydenham in the seventeenth century described true pneumonia under the name *peripneumonia vera* as contrasted with bastard peripneumony—*Peripneumonia notha*, which Huxham in 1764 defined as an obstruction of the lungs by a heavy, viscid, pituitous matter. Was this œdema of the lungs, or (as Dr. Pye Smith states in his article on “Pneumonia” in Dr. Clifford Allbutt's *System of Medicine*) bronchitis?

To Hoffmann belongs the credit of introducing the name *pneumonic fever*, which modern pathology and symptomatology alike justify and approve. To the German school, represented especially by Cohnheim, we owe the term *fibrinous* or *croupous pneumonia*, which means that a coagulable fibrinous exudation takes place on the free surface of the affected lung, that is, into the air-vessels of the affected portion of the organ. *Pleuropneumonia* is a valuable name for the disease, in so far as it emphasises the frequency with which the pleura shares in the morbid process. Lastly, the adjectives “acute,” “sthenic,” “asthenic,” “typhoid,” or “ataxic,” “massive,” and so on, find a place in the terminology of pneumonia, pneumonitis, or lung fever.

In the valleys of the High Alps in Switzerland a severe form of pneumonia in spring is known as “Stich,” or “Alpenstich” (i.e. *stitch*).

The infectiousness of pneumonia was observed by Dünns in 1592. He wrote—“Contagiosum quoque esse inde liquido constabat, quod multi in eadem familiâ corriperentur.” Ravicio, writing at Venice in 1613, uses the words “influsso epidemico maligno con qualche immediato contagio,” of an outbreak “osservato nella terra di Guastalla.” The term *hepatisation*, as applied to one stage of the pathological anatomy of pneumonia, was apparently suggested by Morgagni, who in 1761 used the words—“Pulmones compactam ut in hepate est, substantiam habebant.” Pirri found, in an epidemic at Rome in 1779, “I polmoni degenerare in una sostanza epatica per la sua durezza.”



According to Hirsch, the earliest records of malignant epidemical inflammation of the lungs in Spain, England, Denmark, and North America belong to the eighteenth century. In France and Switzerland many epidemics of pneumonia occurred during that century, the season of nearly all the outbreaks being winter and spring, and the type of the disease being "typhoid" or "ataxic," sometimes "bilious."

The literature of pneumonia grew apace as decade followed decade in the nineteenth century, which also witnessed the elucidation of the ætiology of this *genus* of disease, for pneumonia includes many species. Huxham long ago said: "There are very different degrees, I might say species, of this disease, which demand a particular attention and a method of cure peculiarly adapted to each."

Epidemics of pneumonia occur from time to time. Huxham described an outbreak of a severe type which visited Plymouth in the latter part of 1745 and the beginning of 1746. A pandemic of the disease overran North America between 1812 and 1825. Laennec described an "Epidemic Pneumonia" as occurring among the conscripts of 1814. An outbreak commenced in the Akerhus prison, Christiania, on 18th December 1866, lasting until May 1867. Among an average of 360 prisoners in that period, 62 cases occurred, of which 15 ended in death. Dr. L. Dahl and Professor W. Boeck attributed the outbreak chiefly to overcrowding. To the same cause Dr. Bryson ascribed a very remarkable epidemic of pleuropneumonia which broke out in some ships in the Mediterranean fleet in 1860. Dr. Thoresen of Eidsvold, Norway, met with a local outbreak of "croupous pneumonia" in a single row of cottages near some glassworks in the summer of 1869, when the town was otherwise almost free from the disease. In the spring of 1874 an epidemic of "sewer-gas pneumonia," as it was aptly called, prevailed at East Sheen, Mortlake, London, S.W., in connection with the opening of a ventilator in a main sewer. The cases occurred in a first-class boys' school facing the road under which the sewer ran. During the summer of the same year pneumonia, apparently traceable to sewer-gas poisoning, prevailed in Dublin. It was described under the name of "pythogenic pneumonia" in a paper written conjointly by the late Dr. Thomas Wrigley Grimshaw, C.B., afterwards Registrar-General for Ireland, and by the present writer. In the autumn, winter, and spring of 1874-1875 an epidemic of pneumonia swept over the south-western counties of England (Wynter Blyth). A serious outbreak at Middlesbrough-on-Tees in 1888 formed the subject of an admirable report by Dr. Edward Ballard, Inspector of the Local Government Board, England. The outbreak lasted for 24 weeks, comprised about 1000 cases, and caused 369 deaths. In April and May 1890 there was an outbreak at Scotter, in Lincolnshire, in which 22 deaths occurred among 32 known cases, a mortality of 69 per cent. This epidemic was admirably described by Dr. T. B. Franklin Eminson, medical officer of the Scotter district of the Gainsborough Union, and it was also specially investigated by the Local Government Board inspector, Dr. Parsons, who attributed the outbreak to sewer-air emanations and simple contagion conjointly. In the early spring of 1893 epidemic pneumonia attacked the township of Yeadon, Yorkshire, with a population of 7500. Dr. C. J. Russell M'Lean, D.P.H., medical officer of health for the township, observed 35 undoubted cases, of which 24, or 71·4 per cent, occurred in the months of March and April. There were 13 deaths, the case-mortality being as high as 37·14 per cent. Dr. M'Lean cites two series of cases—3 in each instance—in which the disease was apparently communicated from person to person.

A startling fact as to the frequency and mortality of pneumonia is brought out by Dr. Hector Mackenzie, of St. Thomas's Hospital, who finds that in a population of 30,000,000 in England and Wales, at least 220,830 persons are annually affected with pneumonia, and that 31,950 persons annually die from this disease, the mortality being thus 14·5 per cent of those attacked. In the year 1898 the death-rate from pneumonia in England and Wales was 1323 per million males in the population, and 947 per million females. Tables 20 and 22 in the *Sixty-first Annual Report of the Registrar-General of Births, Deaths, and Marriages in England* (1898) show that the annual death-rate per 1,000,000 from pneumonia among males, which had been as low as 1089 in the year 1881, rose with a bound from 1206 in 1889 to 1731 in 1890, and still further to 1798 in 1891, coincidently with the first epidemic or epidemics of influenza. Similarly among females the annual death-rate, which had been down to 740 in 1881, rose from 848 in 1889 to 1095 in 1890, and to 1165 in 1891. These figures show what a far-reaching effect influenzal pneumonia has upon the death-rate from "pneumonia" in the abstract.

In 1898, out of a total of 82,404 deaths registered in Ireland, 3550 were referred to pneumonia—2074 in males, 1476 in females. That is to say, pneumonia was



responsible for 4·2 per cent of all the deaths, and the death-rate due to it was equal to 1 in 1279·9, or 0·78 per 1000 of the estimated population.

ÆTIOLOGY.—Pneumonia, taking the term in its full acceptation, must be looked upon as an essential or specific febrile disease, strictly analogous to diphtheria, enteric fever, small-pox, or any other of the recognised infective diseases (*Infectionskrankheiten*). It is a microbic infection in which the lungs are the usual, though not the invariable, battleground. In 1899 J. Pelnar reported two cases of pneumococcus sepsis without pneumonia. The first was in an infant three months old, presenting fever and convulsions. The child died, and at the autopsy gastro-enteritis, purulent meningitis of the convexity, and purulent rhinitis were found, cultures from the meningeal pus and the spleen giving the Fränkel-Weichselbaum diplococcus. The second case was that of a woman, aged 22 years, in the puerperium. There was an uncertain diagnosis of scarlet fever with phlegmonous tonsillitis, followed by sepsis and leptomeningitis, cultures from the spleen and from the tonsillar and meningeal pus yielding the pneumococcus. Pneumonia was absent in each case.

The varying phenomena presented in different cases of pneumonia probably depend, as it has been suggested, on a varying virulence of its supposed specific micro-organisms, the affection being regarded as an essential disease analogous to diphtheria, enteric fever, small-pox, or any other infective malady. The micro-organisms which appear to stand in a direct causal relation to pneumonic fever have already been named. But there is evidence to show that the *Diplococcus pneumoniae* and the *Bacillus pneumoniae* are not exclusively concerned in producing inflammation of the lung tissues.

The subject may be considered from both an ætiological—or, we might say, a bacteriological—and a clinical standpoint. Naturally, it is chiefly from the latter point of view that hospital physicians have had opportunities of regarding the question. It is impossible, however, to ignore the bacteriological aspects of the case.

In support of Flügge's statement just quoted, we propose to adduce evidence that the micro-organisms peculiar to erysipelas, to influenza, to tuberculosis, and to enteric fever may one and all give rise to a specific pneumonia or pneumonic fever. So also may Löffler's diphtheria bacillus (the *Bacillus pestis* of Kitasato and Yersin) and the bacillus of malignant anthrax, as well as other pathogenic bacteria. Flügge, for example, says: "We are already acquainted with pneumonias which are caused by aspergillus and actinomyces; it is *a priori* not improbable that also among bacteria there are several other species which can set up pneumonia."

I. ERYSIPELAS.—In the form of this disease which has been called "erratic" or "vagrant erysipelas" (*Erysipelas migrans*)—the *erysipèle ambulante* of French writers—the attack may be protracted for one or two months. In such cases not only every part of the surface of the body, but the whole tract of mucous membranes, and even the lungs and pleuræ, may in turn become affected. Dr. Petèr, of Paris, has drawn attention to the spread of erysipelatous inflammation from the pharynx to the respiratory passages, causing in sequence bronchitis, bronchiolitis (capillary bronchitis), and pneumonia.

In a case observed by the writer at Cork Street Fever Hospital, Dublin, many years ago, the converse of this happened. A man was admitted suffering from *Pneumonia migrans*. After some days a blush of erysipelas showed over one shoulder, and spread thence down the back, with the interesting result that simultaneously the pneumonic symptoms subsided. So great was the impression made upon him by this case that ever since the writer has recognised the



propriety of looking upon *Erysipelas pulmonum* as a distinct species of the great genus Pneumonia.

Grisolle quotes from Serres a case of a patient who had several attacks of pneumonia, each terminating in an attack of erysipelas. Wilson Fox states that he had seen only one such case. The erysipelas appeared three days after complete defervescence, and the resolution of the pneumonia and the subsequent recovery of the patient were greatly protracted.

The teaching of Levy, of Strassburg, that *Streptococcus pyogenes* is an exciter at once of suppuration and of erysipelas, is now generally accepted. This pyogenic bacterium was obtained by Fehleisen from the skin involved in cases of erysipelas in 1883, and by Rosenbach and Passet from the pus of acute abscesses within a year or two afterwards. Sternberg gives the following synonyms for *Streptococcus pyogenes*—*Micrococcus* of erysipelas (Fehleisen), *Streptococcus erysipelatis*; *Streptococcus* of pus, *Streptococcus longus* (von Lingelsheim).

If, then, we admit the identity of the pus-producing streptococcus with that of erysipelas, we at once obtain a key to the occurrence of an acute pneumonia in erysipelas. For this very bacterium—the *Streptococcus pyogenes*—plays a part that is second to none in the production of influenza-pneumonia.

Prof. Hermann Lenhartz, of Hamburg, in his masterly monograph on Erysipelas in Hofrath Prof. Hermann Nothnagel's *Specielle Pathologie und Therapie* (vol. iii. part 3), considers that pneumonia is to be regarded as erysipelalous only when it has arisen in immediate connection with a cutaneous, pharyngeal, or laryngeal erysipelas. Mosny, however, saw a pneumonia appear in the servant of a gentleman ill of facial erysipelas. The attack terminated fatally within two days. At the autopsy a circumscribed broncho-pneumonic area was found, from which pure cultures of streptococci were obtained. These produced a true erysipelas in the ear of a rabbit. Denuce and Schönfeld found the specific streptococci of erysipelas both in the pulmonary capillaries and in the pericardial and pleural exudations in cases of pneumonia occurring in the course of erysipelas.

Waldenburg and Friedreich have emphasised the connection between the so-called "wandering pneumonias" and erysipelas. A serpiginous advance of the pulmonary hepatisation, a marked tendency to involvement of both lungs, the uniform occurrence of marked splenic enlargement, and a frequent coincidence of acute erysipelas and "puerperal fever" caused Friedreich to speak of these cases as "erysipelalous pneumonias."

Lenhartz, in quoting the foregoing authors, while not denying the possibility of primary erysipelalous pneumonias, considers that they are extremely rare. It is, however, certain that such pneumonias may be caused by the *Streptococcus erysipelatis*, and not by the *Diplococcus pneumoniae* of Fränkel, when they accompany or follow an external erysipelas. It may, in his opinion, remain doubtful whether a direct transmission of the germ from the skin to the lungs has caused the pneumonia, or whether the infection has occurred through the blood. In the latter case the erysipelalous pneumonia is to be simply interpreted as a septicæmic manifestation.

II. INFLUENZA.—In the article on this disease (vol. v. p. 275), it was pointed out that the pneumonia of influenza, while producing the ordinary physical signs of acute croupous pneumonia, is often latent in its course or accompanied by a profuse muco-purulent expectoration, with scarcely any rusty sputa. Leyden describes the pneumonias observed by him in influenza as running a peculiar course, severe pain in the side and dyspnoea were rarely noticed, the typical sputum was often wanting, the local process was often atypical—being of a migratory, catarrhal, and of a lobular distribution in many instances. The foremost German authorities on the influenza of 1889-1890 laid stress on the erysipelas-like spread of influenza-pneumonia in the lungs, and on the prominent part which the *Streptococcus pyogenes* seemed to play in its ætiology. The question, however, in such cases is probably one of a secondary infection, for which the attack of influenza merely laid the foundation. Certainly the discovery by Pfeiffer, in 1892, of the *Bacillus influenzae* in the purulent bronchial secretion, and by Canon in the blood of patients suffering from epidemic influenza, must be regarded



as conclusive proof of the existence of a specific primary infection to which all other infections are accidental or secondary.

III. TUBERCULOSIS.—We do not here allude to acute tubercular fever (in which the lungs may escape unscathed) on the one hand, or on the other to the local peripneumonic processes which accompany sporadic depositions of tubercle in the lungs in ordinary catarrhal phthisis. Nor do we refer to those cases in which in the wake of an acute primary croupous pneumonia the wounded lung falls a ready prey to a secondary infection by the *Bacillus tuberculosis*, when this micro-organism finds a fertile soil in the caseating exudation of an unresolved pneumonia. These several conditions are all beside the present question.

Our concern is with acute phthisis, or scrofulous pneumonia, and the so-called acute tuberculo-pneumonic phthisis. Both these affections present consolidations in the lungs of a pneumonic origin, but tuberculisation, as well as pneumonia, exists.

In both these varieties of "Consumption" we have examples of true pneumonia resulting from an infection by the *Bacillus tuberculosis* of Koch.

IV. ENTERIC FEVER.—It is well known that pneumonia is more commonly observed as a complication in enteric fever than in typhus. Murchison noted it in 13 out of 100 cases, and Austin Flint (according to Bartlett) in 12 out of 73 cases. It commonly occurs in the third or fourth week, but may usher in the disease. In this latter case its presence is probably an indication that the enteric fever poison has entered the system through the lungs. It is most commonly a *lobular pneumonia*, but occasionally it occurs under the form of ordinary *croupous pneumonia*.

It is, indeed, true that Eberth points out that anatomical investigations had (up to 1881) afforded no evidence of the admission of the *Bacillus typhosus* through the lungs. With this Gaffky does not agree, for he considers it highly probable—or at least the possibility cannot be contested—that the lungs may occasionally represent the seat of invasion. Eberth himself quotes a case observed by W. Meyer, of Berlin, in which death ensued on the second day of illness. In this case there were found at the autopsy hyperæmia of the lungs, spleen, and kidneys, in the lower portion of the ileum marked swelling of the solitary follicles and Peyer's patches. Microscopical examination revealed a very exceptionally large deposit of Eberth's bacilli in the cells of the submucosa and in the intermediate muscular layers of the intestine. Apparently they were not found in the lungs, notwithstanding their hyperæmic condition.

Even if we take it as not yet proved that infection in enteric fever may occur by way of the lungs, there is no doubt that a close correlation exists between this disease and that variety of acute pneumonia, or pneumonic fever, to which the term "Pythogenic Pneumonia" has been commonly applied since 1875, when the late Dr. Thomas W. Grimshaw, afterwards Registrar-General for Ireland, and the present writer read a paper on the subject before the Medical Society of the College of Physicians of Ireland.

In the eighth volume of the *Johns Hopkins Hospital Reports* (Baltimore, 1900) will be found an analysis and general summary of the cases of enteric fever—829 in number—treated in the medical wards of the Johns Hopkins Hospital from 1889 to 1899. It is from the pen of Professor Osler. Speaking of complications affecting the respiratory system, Dr. Osler says:—

"One may divide pneumonia in typhoid fever into two groups, according to its appearance with the onset, during the course, or towards the termination of the disease." He proceeds: "*Pneumonia at Onset*.—The interesting feature of this form, the so-called pneumo-typhoid of the French, is that the cases may present all the characteristics of ordinary croupous pneumonia, no other diagnosis may be reached, and it may not be corrected until autopsy." He recalls the fact that he had in 1894 given the history of a remarkable case of this character, in which clinically he had no suspicion that there was any other disease than



pneumonia, but the autopsy showed characteristic enteric fever. He reports a very similar case, occurring in a man aged 68, who died within the first forty-eight hours without any suspicion having been entertained that he had enteric fever. The autopsy showed the lesions of this disease. Again, in the case of a woman aged 26, who was admitted on November 11, 1898, complaining of cough, headache, and pain in the back, the symptoms remained chiefly pulmonary for nearly eleven days, and Dr. Osler and his colleagues did not recognise the case as one of enteric fever. There was no leucocytosis at first. On November 13 there were 7600 white corpuscles; on November 22, 23, and 24, they increased to 22,000; on November 28 there were 12,500. The patient had no diazo reaction throughout. The Widal test was applied repeatedly, but it was not until November 24 that it could be said to be fairly positive. Then on the 26th a crop of rose spots and the general features clinched the diagnosis.

As regards enteric fever, the influence of season and weather in determining pneumonic trouble is, no doubt, considerable; but it cannot be accepted as paramount or exclusive. And, indeed, it is reasonable to suppose that in some cases, at all events, the pneumonitis is directly due to a localisation of the specific poison of enteric fever in the lung, whether that poison be the *Bacillus typhosus* itself, or a toxin derived therefrom.

V. ANTHRAX.—“That man may be infected with anthrax by way of the respiratory organs,” writes Sternberg, “seems to be well established. In England the disease known as ‘wool-sorters’ disease’ results from infection in this way among workmen engaged in sorting wool, which is liable to contain the spores of the anthrax bacillus when obtained from the skin of an animal which has fallen a victim to this disease. That infection occurs through the lungs is shown by the fact that these organs are first involved, the disease being, in fact, a pulmonic anthrax.”

That, in infective diseases in general, infection may occur through the mucous membranes of the respiratory tract, has been demonstrated (according to Sternberg) by several bacteriologists—especially by Buchner, who caused mice and guinea-pigs to breathe an atmosphere containing in suspension a powder consisting of dried anthrax spores mixed with lycopodium powder, or pulverised charcoal. In a series of 66 experiments, 50 animals died of anthrax, 9 of pneumonia, and 7 survived. Microscopical examination of sections and culture experiments showed that the lungs were extensively invaded.

VI. EPIDEMIC CEREBRO-SPINAL MENINGITIS.—The *Diplococcus intracellularis meningitidis* of Weichselbaum occasionally produces a specific pneumonia. In a report on an epidemic of cerebro-spinal meningitis which prevailed in Boston, Massachusetts, and the neighbouring towns, from the spring of 1896 to that of 1898, published by the Massachusetts State Board of Health, Councilman, Mallory, and Wright mention 8 cases in which the diplococcus caused pneumonia, in addition to which there were 2 cases of croupous pneumonia with pneumococci, 7 of broncho-pneumonia, and 13 cases of congestion with œdema.

VII. PLAGUE.—The most fatal form of this dread disease is that in which it declares itself as a true pneumonia, albeit more frequently of a disseminated kind (broncho-pneumonia), of a lobular rather than of a lobar type. It is not merely deadly, but dangerous, because of the vast crowds of bacilli which are found in the expectoration, whereby the infection may be widely diffused.

During the epidemic of plague in Bombay in 1897, Captain L. F. Childe, I.M.S.,<sup>1</sup> observed that, concomitantly with the increase of mortality from the bubonic form of the disease, deaths from pneumonia multiplied *pari passu*. In the pneumonic

<sup>1</sup> Report to the Plague Research Committee in Bombay.



form of plague, according to Childe, the only marked evidences of disease are found in the lungs; the lymphatic glands and other organs are scarcely affected at all. In these cases there was general engorgement, with considerable œdema and a reddened condition of the bronchial mucous membrane. A number of pneumonic patches, varying in size from a pea to an egg, were found scattered through the lungs. These patches had the appearance of the first and second stages of lobular pneumonia, but no patches were found which had passed on to the third stage of softening and breaking down. Petechial hæmorrhages were usually forthcoming on the surface of the lung. The bronchial glands were either enlarged, swollen, œdematous, soft, and distinctly engorged, or else they were small and of the usual appearance—perhaps a little engorged.

In cases of plague pneumonia Childe states that the bacilli of plague were seen in abundance in the pneumonic areas. They could be found in profusion amongst the catarrhal epithelial cells and leucocytes which filled the alveoli and terminal bronchioles, as well as among the blood corpuscles of the alveoli into which hæmorrhage had occurred. Similarly in the lungs of non-pneumonic cases they could be seen, but in far fewer numbers, and mostly where small hæmorrhages had taken place into the alveoli.

Dr. James Cantlie says that pneumonic plague is usually ushered in by a rigor, dyspnoea, cough, a high temperature, and marked prostration. The sputum is profuse, of a watery character at first; it may be tinged with blood from the onset, but more usually blood does not appear until the illness has lasted twenty-four hours. The pulse is soft and compressible from the first, and cardiac distress is considerable. Pneumonic patches—lobular pneumonia—occur in either lung and at any portion of the lungs, but are more usually met with in the lower lobe and towards the posterior aspect. Moist sounds, especially at the base, and bronchial rhonchi are to be heard, but all the signs and symptoms are peculiarly evanescent, and a patch of pneumonic consolidation, dull to-day, clears up to-morrow when another part of the lung becomes affected. Delirium, frequently of a violent type, is present; but before death coma supervenes, preceded, it may be, by a muttering delirium. The pneumonic variety of plague is a most rapidly fatal form of the disease, the patient usually dying on the third or fourth day.

In an article on "Pneumonia and Broncho-Pneumonia," published in February 1900, the editor of *The Practitioner* (Mr. Malcolm Morris) wrote as follows:—"Certain pathologists, without contesting the intervention of the pneumococcus in the majority of cases of lobar pneumonia, consider that this microbe is not always *en cause*; that other micro-organisms can give rise to this disease. To unity in cause, they oppose plurality (Weichselbaum, Jürgensen, Finkler). Their opinion is founded on this consideration, that in a pneumonic focus other pathogenic microbes than the pneumococcus have been found; that these microbes introduced into the lung of animals have given rise to inflammatory lesions."

In contradistinction to these views, which we venture to adopt from our own clinical experiences, Netter says dogmatically: "There is no pneumonia without pneumococci." He also states that "the constant presence of the pneumococcus of Talamon-Fränkell in lobar pneumonia, and the plurality of microbes causing broncho-pneumonia, are the distinctive elements which bacteriology permits us to recognise in the history of pulmonary inflammations." According to Netter, the pneumococcus of Talamon and Fränkell is the specific microbe of *lobar* pneumonia, but he will surely admit that the pneumococcus plays a leading part in the causation of pneumonia in children, yet how seldom is its distribution lobar. Nor is the lung by any means the only seat of a primary localisation of the pneumococcus. In 121 post-mortem examinations Netter himself found the primary localisation to lobar pneumonia in 65·95 per cent, broncho-pneumonia and bronchiolitis in 15·85, meningitis in 13, purulent pleurisy in 8·53, otitis media in 2·44, endocarditis in 1·22, and abscess of the liver in 1·22 per cent.



## PREDISPOSING CAUSES

1. *Town life* increases both the prevalence of and the mortality from pneumonia. The Registrar-General for Ireland (Mr. Robert Matheson, LL.D., M.A.) has kindly furnished tables showing the number of deaths from pneumonia by ages and sexes registered in the whole of Ireland and in the Dublin Registration District respectively during each of the five years 1894-1898, together with the average deaths from pneumonia for the same period. From these tables it appears that the average annual death-rate per 10,000 of the population from pneumonia throughout Ireland in the quinquennium 1894-1898 was 7·8. The corresponding figure for the Dublin Registration District was 14·4. So that practically the pneumonic death-toll was twice as heavy in the Irish metropolis as it was throughout the country at large. In other words, pneumonic affections kill twice as many of the population in a large town compared with the open country.

2. *Age*.—From the same tables we learn that the average annual deaths from pneumonia in all Ireland during the five years 1894-1898 were 3574. Among these were 395 deaths of infants under one year of age, 245 of children aged 1 year, 103 of children aged 2 years, 65 of children aged 3 years, and 39 of children aged 4 years—in all 847 deaths of children under 5 years of age, or 23·7 per cent. Between the ages of 5 and 10 years the deaths were 111; from 10 to 15 years, 71; from 15 to 20, 104; from 20 to 25, 124; from 25 to 35, 259; from 35 to 45, 335; from 45 to 55, 437; from 55 to 65, 571; from 65 to 75, 463; from 75 to 85, 215; from 85 to 95, 35; and at 95 years of age and upwards, 2.

3. *Sex* plays an important part in the ætiology of pneumonia. In England and Wales, in 1898, 1323 males out of every million died of pneumonia, while the corresponding number of females was only 947. Three-fifths of the deaths from this disease registered in Ireland in the five years 1894-1898 inclusive were among males, two-fifths among females. The exact numbers were—males, 2111 deaths, or 59 per cent; females, 1463 deaths, or 41 per cent; total deaths, 3574. Sex, however, is rather an accidental than a positive factor in the ætiology, for it is only after the age of 20 years that the disproportion in the deaths among the sexes becomes extreme. Under 1 year old, indeed, out of 395 deaths, only 166 were of females, whereas 229 were of males, the percentages being—females, 42, males, 58. Between the ages of 1 and 2 years the respective deaths were—males, 135; females, 110; total, 245; or, in percentages, males, 55; females, 45. Then comes a change—between 2 and 3 years the sexes die of pneumonia in equal numbers—males, 52; females, 51; total annual deaths, 103. Between 3 and 4 years the numbers are—males, 30; females, 35; total, 65; between 4 and 5—males, 18; females, 21; total, 39. The above figures give 847 deaths yearly, of which 464, or 55 per cent, occur among males, 383, or 45 per cent, among females. But between 5 and 10 years, 57 females die of pneumonia for every 54 males; and between 10 and 15 years, 39 females for every 32 males. Taking these ten years—5 to 15—together, we find that of 182 yearly deaths only 86 occur among males, but 96 among females, the percentages being—males, 47; females, 53. After puberty, *i.e.*, 15 years, the sexes draw more and more widely apart as regards the deadly influence upon them of pneumonia, the balance being altogether against the male sex. The explanation is obvious. After the critical age of fifteen males are more exposed to the other predisposing causes of pneumonia, including over-exertion, over-heating, vicissitudes of atmospheric temperature, wet and cold, and, above all, alcoholic intemperance and injury.



4. *Season*.—Contrary to what one might *a priori* imagine, pneumonia is not so much a disease of winter as of spring, and of late spring especially.

August Hirsch, Professor of Medicine in the University of Berlin, after pointing out that pneumonia, even in its narrowest acceptation of fibrinous or so-called croupous pneumonia, is an anatomical term that includes several inflammatory processes differing from one another in their ætiology, goes on to observe that the prevalence of the malady depends very decidedly upon certain influences of season and weather. He gives an elaborate table of percentages of pneumonic prevalence in the several months at a large number of places in Europe and America. According to this table, the largest number of cases falls in the months from February to May; the smallest number in the period from July to September. Taking the average for all the places mentioned in the table, it appears that 34·7 per centum of the patients were attacked in spring (March to May, inclusive); 29·0 in winter (December to February); 18·3 in autumn (September to November); and 18·0 in summer (June to August). The combined percentage for winter and spring is 63·7; that for summer and autumn is 36·3. If the number of cases in summer be taken as 1, then autumn has 1·02, winter 1·6, and spring 1·9. Nearly all the recorded epidemics of pneumonia have occurred in winter and spring. From the foregoing considerations, Hirsch confidently concludes that the origin of the malady is dependent on weather influences proper to winter and spring; and more particularly on *sudden changes of temperature and considerable fluctuations in the proportion of moisture in the air*. He holds that any exceptionally large number of cases of "inflammation of the lungs" at the other seasons, more especially in summer, has coincided with the prevalence of the same meteorological conditions phenomenally at that season.

Netter thinks that the maximal prevalence of pneumonia in March, April, and May is due to an increased virulence of the pneumococcus during those months.

In a paper on "Pythogenic Pneumonia," published in 1875 by the late Dr. Grimshaw and the writer, will be found a table, compiled from the returns of the Registrar-General for Ireland, which shows the number of deaths from bronchitis and pneumonia registered in the Dublin Registration District in each quarter of the nine years, 1865-1873, inclusive. According to that table, of every 100 deaths from bronchitis, 44 on the average occurred in the first quarter of the year, 22 in the second, only 10 in the third, and 24 in the fourth quarter. Thus, the mortality from bronchitis was twice as great in the first as it was in the second quarter, and more than four times greater in the first than in the third quarter.

Very different were the facts as to pneumonia—of every 100 deaths from this disease, 32 on the average occurred in the first quarter, 27 in the second, 16 in the third, and 25 in the fourth quarter. The mortality from pneumonia was only *one-fifth* greater in the first than in the second quarter, and only twice as great in the first as in the third quarter. The extreme winter fatality of bronchitis and its low summer fatality were equally wanting in the case of pneumonia.

A careful analysis of the weekly returns of the Registrars-General of England and Ireland for ten years ending with 1885, and of the same returns for the year 1886, brings out a similar remarkable contrast between bronchitis and pneumonia, as to the time of year when these diseases are respectively most prevalent and fatal in London and Dublin.

In both cities bronchitis falls to a very low ebb in the third, or summer quarter of the year (July to September, inclusive), when only 12 per centum of the deaths annually caused by this disease take place in Dublin, and only 11 per centum in London. In the last, or fourth quarter (October to December, inclusive), the percentage of deaths from bronchitis rises to 27 in Dublin and to 30 in London. The maximal mortality occurs in the first quarter (January to March, inclusive), when it is 38 per centum in both London and Dublin. In the second, or spring quarter (April to June, inclusive), the deaths from bronchitis declined to 23 per centum in Dublin and to 21 per centum in London.

The mortality from "pneumonic fever" is very differently distributed throughout the year. In the summer quarter more than 14 per centum of the deaths yearly referable to this disease are recorded in Dublin, and more than 15 per centum in London. In the first quarter the figures are—Dublin, 31 per centum; London, 31 per centum; in the second quarter they are—Dublin, 30 per centum; London, 26 per centum; in the fourth quarter they are—Dublin, 24 per centum; London, 28 per centum.

From these numerical results it, therefore, appears that the fatality and (indirectly) the prevalence of pneumonic fever from season to season do not



correspond with the seasonal prevalence and fatality of bronchitis. The latter disease—be it of primary or secondary origin—increases and kills in direct relation to the setting in of cold weather, with excessive relative humidity and increased and frequent precipitation in the form of rain, snow or sleet, and hail. It subsides in prevalence and fatality with the advance of spring and the advent of summer.

Pneumonic fever, on the other hand, increases less quickly in winter, and remains more prevalent and fatal in spring and summer than bronchitis; its maximal incidence coincides with the season of dry, harsh winds and hot sunshine in spring, when also the relative humidity is low, precipitation is scanty, while the diurnal range of temperature is extreme.

5. *Intemperance* is generally credited with increasing the predisposition to pneumonia. It certainly increases the risk to life caused by this disease. Dr. Hector Mackenzie, however, states that it has not been clearly proved that alcoholism makes an individual more susceptible to pneumonia. There was a clear history of alcoholism in about 8 per cent of 1392 cases treated at St. Thomas's Hospital, and in 9·8 per cent of the cases reported on by the Collective Investigation Committee of the British Medical Association.

6. *Occupation*.—In a letter to the Registrar-General of Births, Deaths, and Marriages in England, on the mortality of males engaged in certain occupations in the three years 1890-92, Dr. John Tatham, M.A., states that out of every 1000 deaths among males aged between twenty-five and sixty-five years, 107 were referred to "pneumonia." The "comparative mortality figure" for this disease varied from 257 for innkeepers in the industrial districts, 251 for general labourers in the same districts, 249 for coal-heavers, 248 for those employed in nail, anchor, chain, and other iron and steel manufactures, and 246 for hotel servants in London, to 45 for clergymen, 55 for lawyers, 93 for physicians, surgeons, and general practitioners, but only 32 for artisans, 36 for farmers, and 43 for schoolmasters. In the higher figures Dr. Arthur Newsholme thinks that the effects of dust and of alcoholism are probably combined.

### INCUBATION

Definite references to the duration of the incubation period in pneumonic fever will be found at page 41 of a pamphlet on *Epidemic Pneumonia at Scotter*, by T. B. Franklin Eminson, published by H. Kimpton (London) in 1892; and in Dr. Parsons' report on the outbreak in question, published in the supplement to the *Twentieth Annual Report of the Local Government Board*, 1890-91 (pages 95 *et seq.* of the supplement).

Mr. Eminson says: "It is the general opinion that the incubation period is very short—a few hours to a few days—and this agrees with the experience of other epidemics; indeed, it has been said that there is frequently no incubation period properly so called, the patient falling ill very soon after exposure." Mr. Eminson narrates two cases in which the interval between exposure to the cause of the disease and its onset was 60 and 24 to 48 hours respectively. These periods, however, were only highly probable, by no means positively certain. Dr. Parsons says in his report: "The length of the period of incubation is doubtful, but it appears to be short, from a few hours to two days (assuming the disease to have been contracted at the time and in the manner locally supposed)." Dr. C. J. Russell M'Lean, in a paper on *Epidemic Pneumonia at Yeadon, Yorkshire*, in 1894, mentions three cases of pneumonia, in which a wife took ill on the fourth day of her husband's illness, their son-in-law falling sick five days later; all three cases proved fatal. In December 1899 the writer met with a somewhat similar instance—a sister falling ill on the fourth or fifth day of her brother's attack, and both dying of epidemic pneumonia.

The following instructive series of cases was reported in *Lyon médical*, April, 28, 1889:—A shopkeeper's child was convalescing from pneumonia when a servant



boy developed the disease. He was removed to hospital, another boy taking his place, wearing the same suit of livery, and sleeping in the same bed as his predecessor. Two days afterwards he sickened with pneumonia. A third boy was now engaged. He slept for two nights with the second boy, and thirty hours later fell ill of acute pneumonia.

It is interesting to observe that the life-history of the *Diplococcus pneumoniae* of Fränkel would almost preclude an incubation stage of more than five or six days at the outside. The virulence of this specific micro-organism, at all events when cultivated in solid media, is spent at the end of seven days, curiously enough the very period within which acute pneumonia is limited by its sharp and critical termination.

### CLINICAL FEATURES

In a large number of cases the prodromata and early symptoms of pneumonia differ in no way from those of any other infective fever. They are referable to the nervous system rather than to the lungs, and so betoken a general or constitutional poisoning as well as a mere local pulmonary infection.

In such instances the actual outburst of pneumonia fever is preceded by hours, or even days, of impaired health, signalised by languor, indigestion, loss of appetite and constipation, headache and dull pains in various parts of the body, alternate chills and flashes of heat, together with a general disinclination to work or amusement. To convey an idea of this vague departure from health the expressive though clumsy word "all-overishness" has been coined, but the state is better described by the more subtle French term "malaise." Andral relates a case in which the general symptoms of headache, debility, dulness of the intellectual faculties, flushed face, injected eyes, and frequent pulse, appeared at least six days before the physical signs of pneumonia could be detected.

Not infrequently the onset of the disease may be stormy in its suddenness and impetuosity. A violent shivering fit, a racking headache and delirium, nausea and vomiting, a thickly coated tongue and foul breath, may, in such a case, suggest an attack of epidemic meningitis, or typhus, or acute gastritis, or enteric fever. All at once physical signs of a rapidly advancing consolidation of the lung—probably of the right apex—declare themselves, the general state of the patient simultaneously improves, and the attack may henceforth run a normal course.

But in more typical instances pneumonia is ushered in with sharp, lancinating chest-pain, quickened, shallow breathing, cough, which is either constant or paroxysmal, hard and dry, as well as suppressed because of the pain it causes. This pain, when acute, is certainly often due to pleuritis, but when dull it may also indicate intense hyperæmia of the portion of lung which is the destined battlefield of the attack. Its usual situation is a limited area (which is also tender on pressure) near either nipple, but sometimes it is felt in the side (infra-axillary region), or in either hypochondrium, or even in the abdomen. In the last-named case it is very misleading, suggesting a gastric hepatitis or intestinal lesion rather than a lung attack. After a couple of days the pain subsides or disappears, as the movements of the affected lung gradually lessen, owing to consolidation due to coagulation of the exudation in the air cells.

Within the first few hours of the attack a very perceptible difference of movement on the affected and sound sides of the chest may be observed. The expansion lessens—it may be to vanishing point—on the affected side, whereas it increases considerably on the other, the sound lung working more and more energetically in response to the strain put upon it through the maiming of its fellow.



Many cases of acute pneumonia begin with a shivering fit or chills, which imply that toxins have already reached the central nervous system by absorption from microbic growth in the pulmonary air cells. The more intense this initial rigor the more deadly the poisoning. A sense of profound

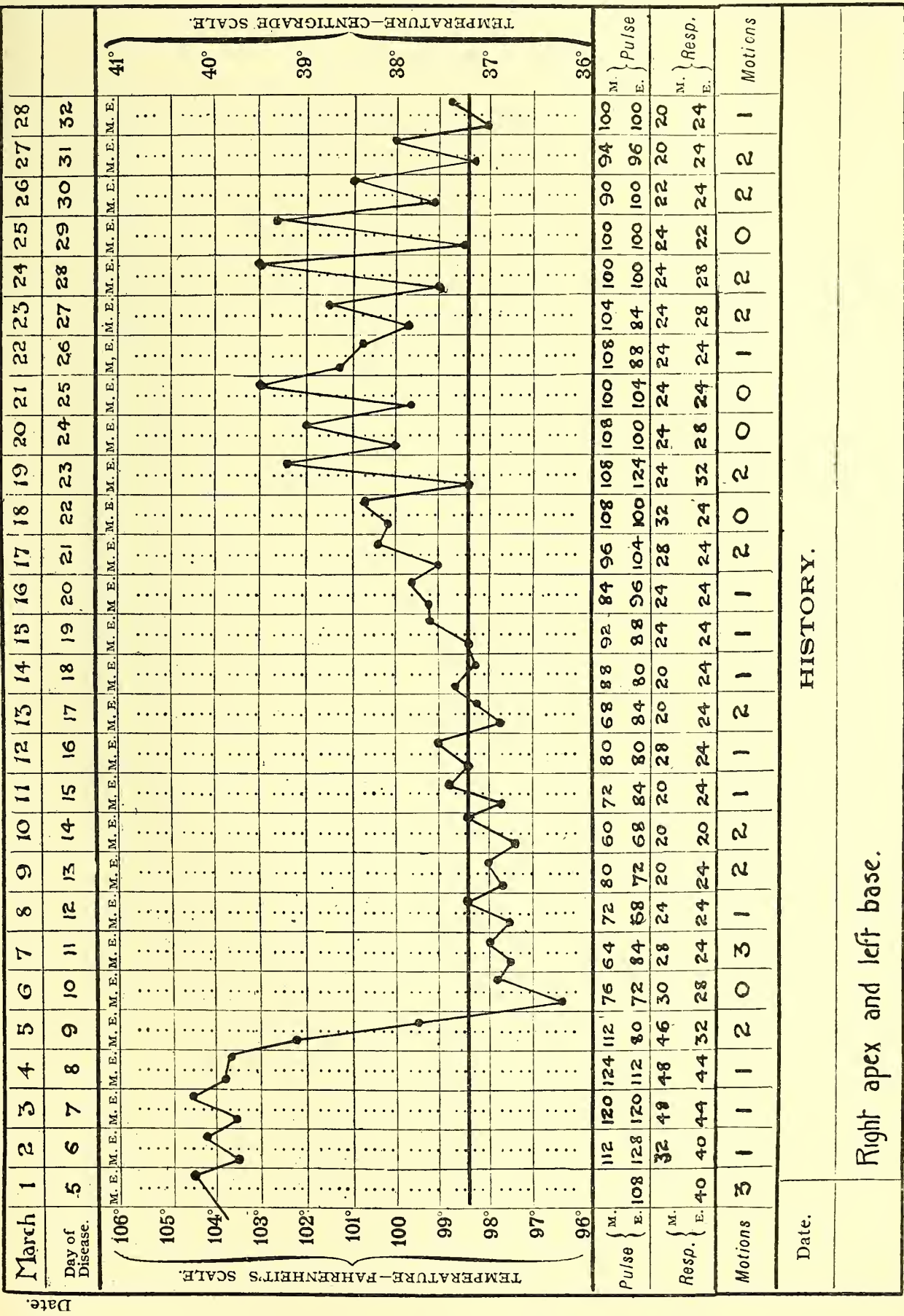


CHART 1.—Acute Right Apical Pneumonia followed by a secondary Septicemic Attack.

depression is apt to follow the shivering fit, or other nervous symptoms, such as headache, delirium, in children even convulsions or vomiting, with constipation or diarrhoea, quickly declare themselves. Another nerve symptom is the development of crops of herpes on or near the lips, the nostrils, the



cheek, the chin, or the jaws—this *herpes labialis* or *faciei* consists of groups of vesicles on a more or less hyperæmic base. The contents of the vesicles are at first serous, but may become purulent. They afterwards dry up and crust or scab, and the scabs finally fall off without leaving a scar. The development of herpes is usually looked upon as a favourable sign. It occurs in about two-thirds of the cases of acute pneumonia, but its apparent dia-

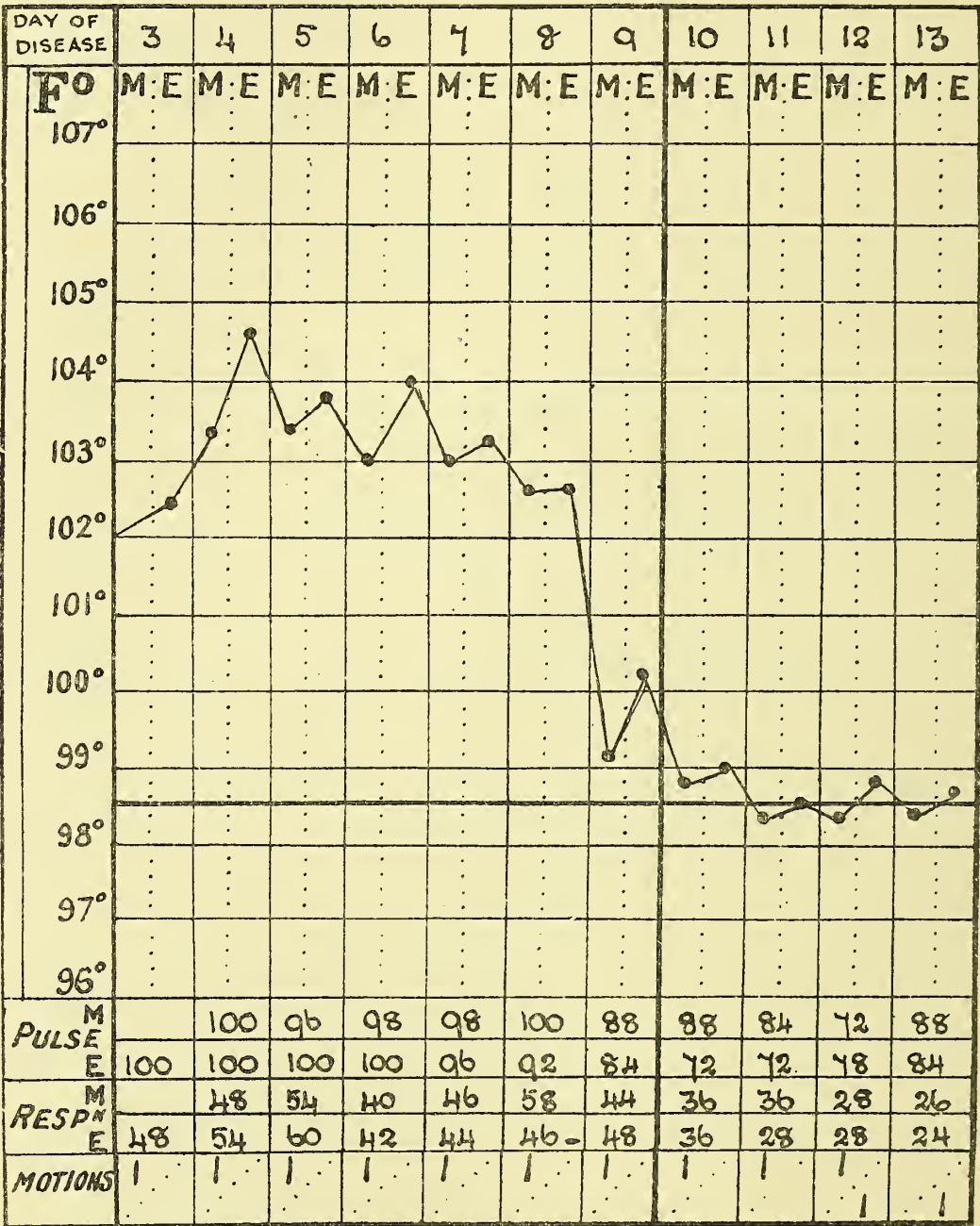


CHART 2.—Acute Pneumonia terminating by Crisis. Patient, a male aged 18.

gnostic value is minimised by its common appearance in simple fevers and ordinary catarrhal attacks. Geissler has estimated the mortality in herpetic cases at 9·3 per cent, in non-herpetic cases at 29·3 per cent.

The aspect of a patient in the early stage of pneumonia is flushed and feverish, often anxious. A deep hectic blush settles upon one or other cheek—the “malar flush.” The skin is dry and hot, almost burning the observer’s hand with its pungent heat. This is the so-called *calor mordax*, common to pneumonia with scarlatina (Addison) and the hot stage of intermittent fever. According to Wunderlich, of Leipzig, this pungent heat of skin belongs to those febrile diseases in which the high temperature depends chiefly upon increased production of heat, and in which the hand of the observer is therefore less able to place itself in equilibrium with the temperature of the patient’s skin, because the continual over-production of



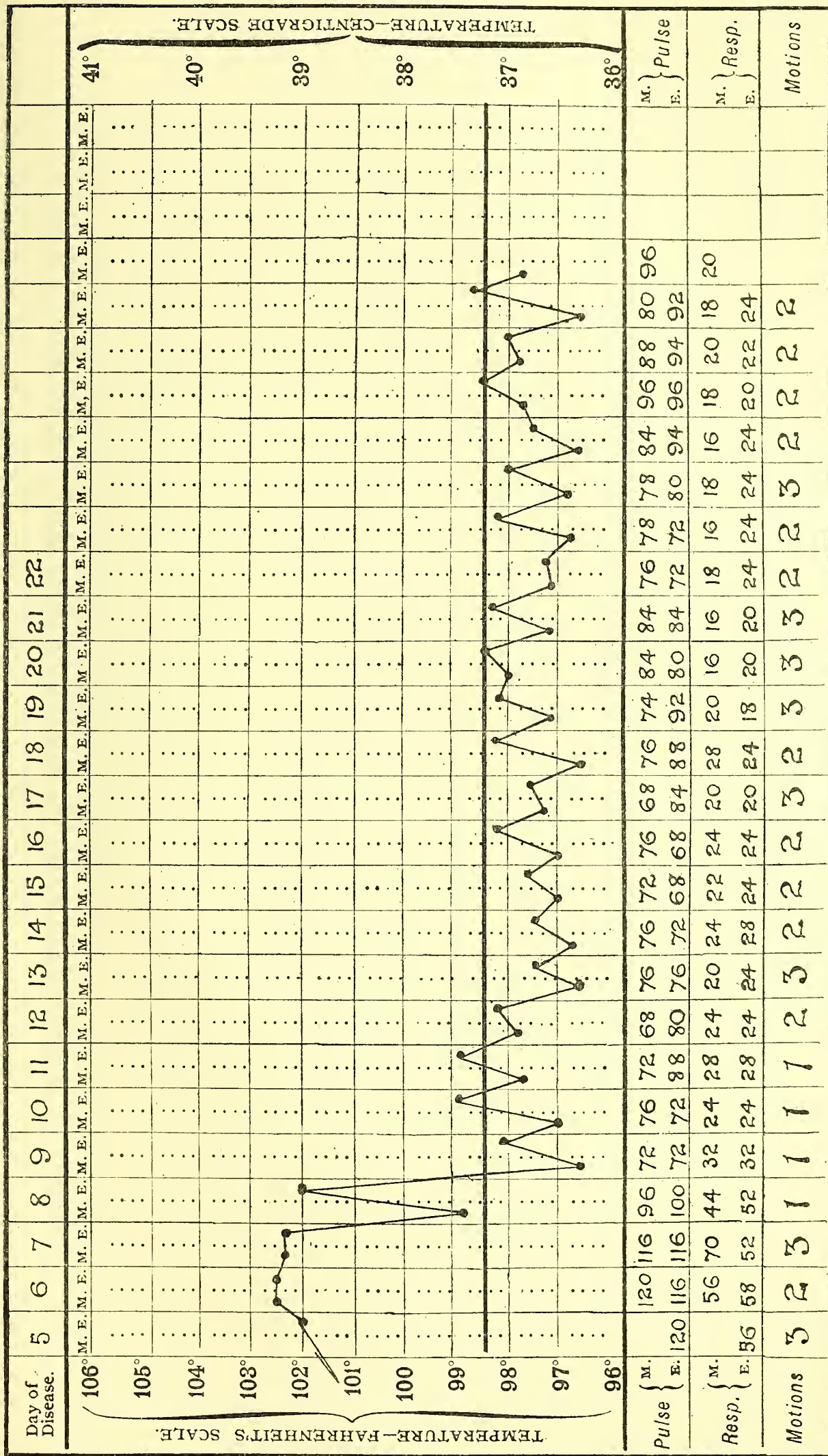


CHART 3.—Acute Pneumonia terminating by a Halting Crisis. Patient, a man aged 18. Right Base affected.



body-heat (exalted thermogenesis) compensates for the heat lost by conduction. The relaxed or dilated state of the arterioles in pneumonia may also be a cause of *calor mordax*.

After an initial rigor or rigors, the thermometer rises briskly and in a few hours to 102° or upwards. It continues to rise until it reaches 104° F., or in severe cases 105·8° or more. For some days temperature hovers near these figures according to the intensity of the case, showing, it may be, daily fluctuations of one or two degrees, the remissions occurring usually in the morning, the exacerbations in the afternoon or at night. Often a sudden and final rise of temperature to a fastigium immediately precedes the abrupt and critical defervescence so characteristic of pneumonic fever, and which may happen at any time between the fifth and the eighth days of the attack. In other instances the fastigium is reached early, and a daily descent of temperature precedes the final crisis. Such is the continuous or subcontinuous type of pneumonic fever.

But other types present themselves, particularly in children, whilst the temperature chart is remittent or relapsing in character—a peculiarity very suggestive of a lobular rather than a lobar pneumonia, or of what has been well called a *pneumonia migrans*, wandering pneumonia, which, erysipelas-like, invades region after region of the pulmonary parenchyma. In this form of the malady affected portions of lung clear up or resolve with marvellous rapidity, while other portions are passing into the earlier stages of hyperæmia, congestion, or consolidation, as the case may be. It is possible in such a case to study the physical signs of each and every stage of an acute pneumonia at once.

Very early in the attack the rate of breathing becomes quick, shallow, and laboured (*dyspnœa*). The respirations number 40 to 48, or even 60 or upwards, per minute, and this too when the pulse-rate may not exceed 120. The normal ratio of one act of respiration to four beats of the pulse is therefore lost in pneumonia, and so commonly as to constitute an important diagnostic sign of the disease. The difficulty of breathing is ever and anon increased by a short hacking cough, which the patient strives, though often in vain, to suppress because of the torture it causes by shaking the affected side, or by putting the affected lung and pleura on the stretch. At first the cough is dry, or unattended by expectoration. But, in most cases, sputa soon begin to come up, often with much difficulty and suffering. These sputa may be simply bronchitic in character, colourless, frothy, liquid, and consisting of mucus, epithelium, and young cells. Occasionally they at first consist almost entirely of pure blood. There is a true hæmoptysis.

Usually, however, there is no mistaking the expectoration of acute lobar pneumonia. It is rust-coloured, semi-clear or transparent, and so viscid as to cling to the bottom of the spitting cup even when it is turned upside down. Examined microscopically this very characteristic sputum is found to consist of a network or mesh of coagulated fibrinous exudation, in which are entangled red blood corpuscles, giving it the rusty colour, altered epithelium from the air vessels and bronchioles, and many leucocytes. Such are the histological elements in the sputum of acute pneumonia of the croupous or fibrinous form. Occasionally the colour of the expectoration is greenish yellow instead of rusty. This is the so-called “green-gage” sputum, because of its resemblance to the well-known preserve.

In certain unfavourable cases the sputa are thin or watery in consistence, dark coloured, and offensive, resembling liquorice juice or prune juice. This is a bad sign. So also is a complete absence of expectoration, which may result from palsy of the bronchi and retention, or from the so-



called “massive pneumonia,” in which lung-cells and bronchioles alike are plugged with firm fibrinous coagula, which no effort of coughing is able to dislodge.

Coincidentally with the beginning of the stage of resolution the expectoration undergoes a complete change. It loses its sticky, viscid character, and its rusty colour gives place to yellow and finally white. It is coughed up more easily and in abundance. While yellow it is airless, sinks in water, and forms rounded, coin-like masses, whence it is called “nummular” (Lat. *nummus*, a coin). Finally, it becomes less and less purulent, and more frothy and mucoid. These changes are an outward sign of those which are taking place in the affected portion of lung. The fibrinous exudation has undergone fatty degeneration, has become emulsified, and is being in part absorbed, in part coughed up. Under the microscope the sputum in this stage is seen to consist of disintegrated young cells, fat granules, pigment, and fragments of fibrinous casts of the alveoli and terminal bronchioles.

In a final stage the sputum becomes a mere frothy mucus, such as is spat up in an ordinary bronchial catarrh.

During, and sometimes after the conclusion of, the pyrexial period, nervoussymptoms, such as sleeplessness, delirium, and wandering may present themselves, particularly if the apex of the lung is the special seat of the disease, or if the patient has been intemperate. In the latter case an acute attack of delirium tremens may supervene. A certain amount of rambling of mind at night is common, but the appearance of such a symptom by day is of the utmost import, and often foretells a fatal issue. On the whole there is less tendency in acute pneumonia to cerebral symptoms than in typhus, or some of the other acute infections; the decided localisation of the poison in the lungs apparently relieves the nervous system, which often suffers severely when such a localisation does not take place or is of feeble intensity.

In a large number of cases pneumonia ends as suddenly as it began, so far at least as the constitutional symptoms are concerned. The storm and stress of a tempestuous fever give place to the calm and comfort of returning health. So periodic and rapid is the defervescence, that of the pneumonic patient it may often be

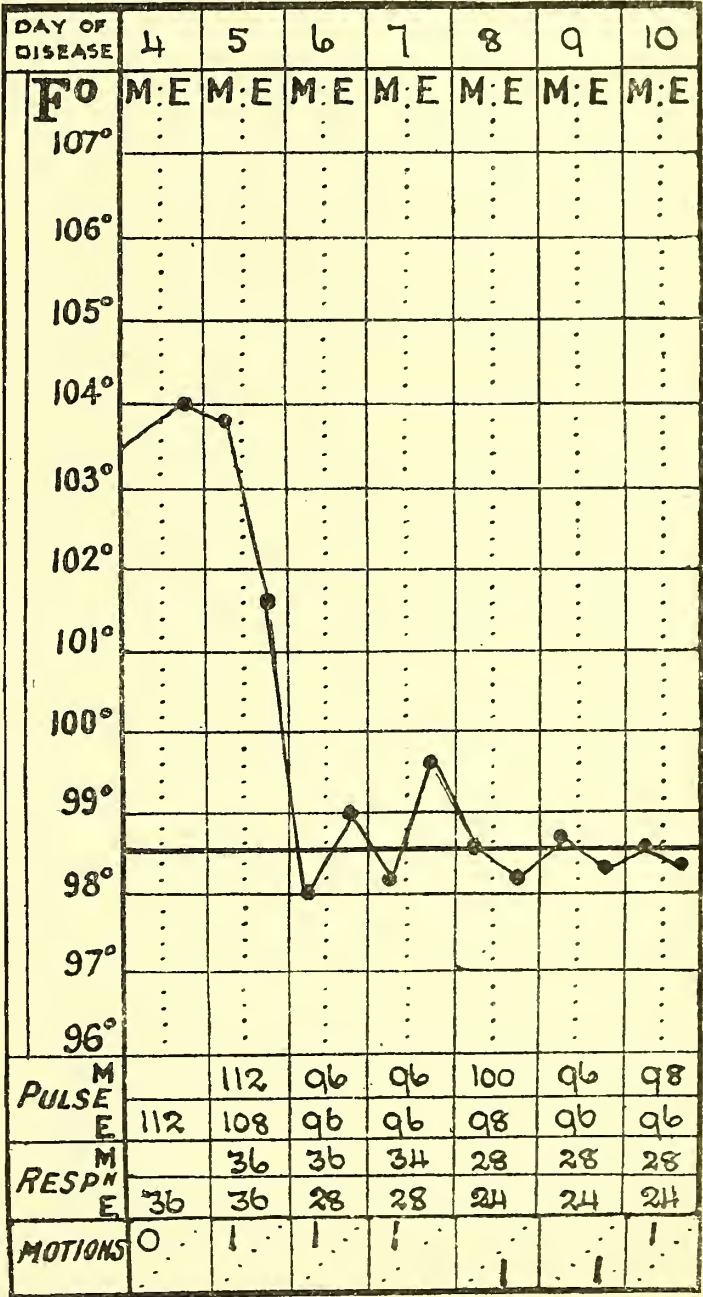


CHART 4.—Crisis in Pneumonia on 5th Day. Patient, a woman aged 22.



said that “at such an hour the fever left him.” The physical signs, however, persist for a longer or shorter time after crisis, for the clearing or “resolution” of a consolidated lung or pulmonary lobe takes time. The fibrinous exudation undergoes a fatty degeneration, and at the same time liquefies. The resulting albuminous and fatty emulsion is then partly absorbed, partly expectorated.

Two notable departures from the cyclic course of pneumonia just described now and then present themselves. In a certain though small proportion of cases, after a threatening onset and one or two days of moderate, or it may

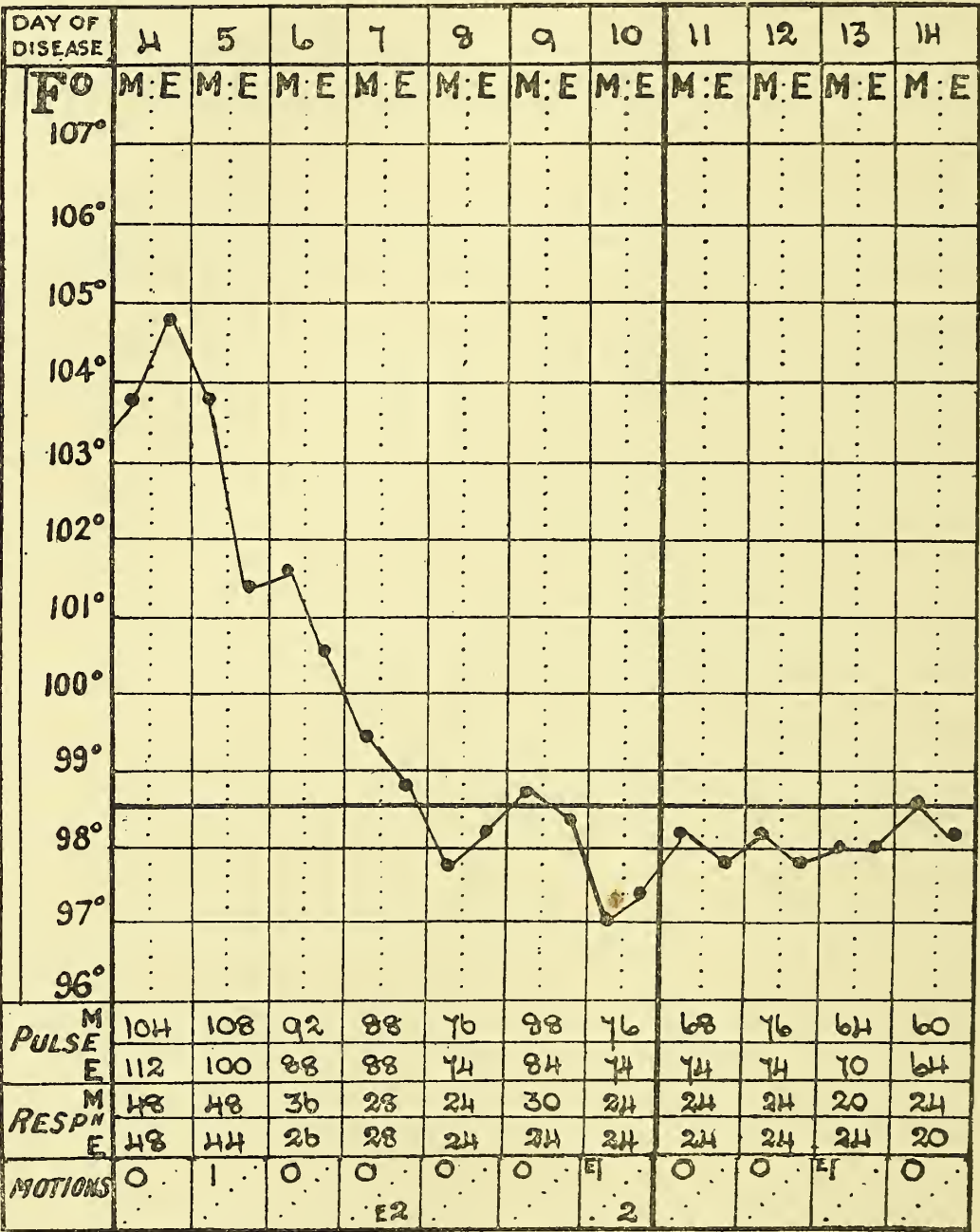


CHART 5.—Protracted Crisis in Acute Pneumonia.

be high fever, a sudden change for the better takes place with a brisk fall of temperature, a moist perspiring skin, and a critical sleep. The attack has aborted. It is an example of *masked* or *larval* pneumonia. Professor Osler, of Baltimore, thus describes it:—“A patient may have the initial symptoms of the disease, a slight chill, moderate fever, a few indefinite local signs, and herpes. The whole process may last for only two or three days; some authors recognise even a one-day pneumonia.” In arriving at a diagnosis in such a case, the epidemic prevalence of the disease at the time, and the occurrence of undoubted pneumonia in the same house or family, should be taken into account.



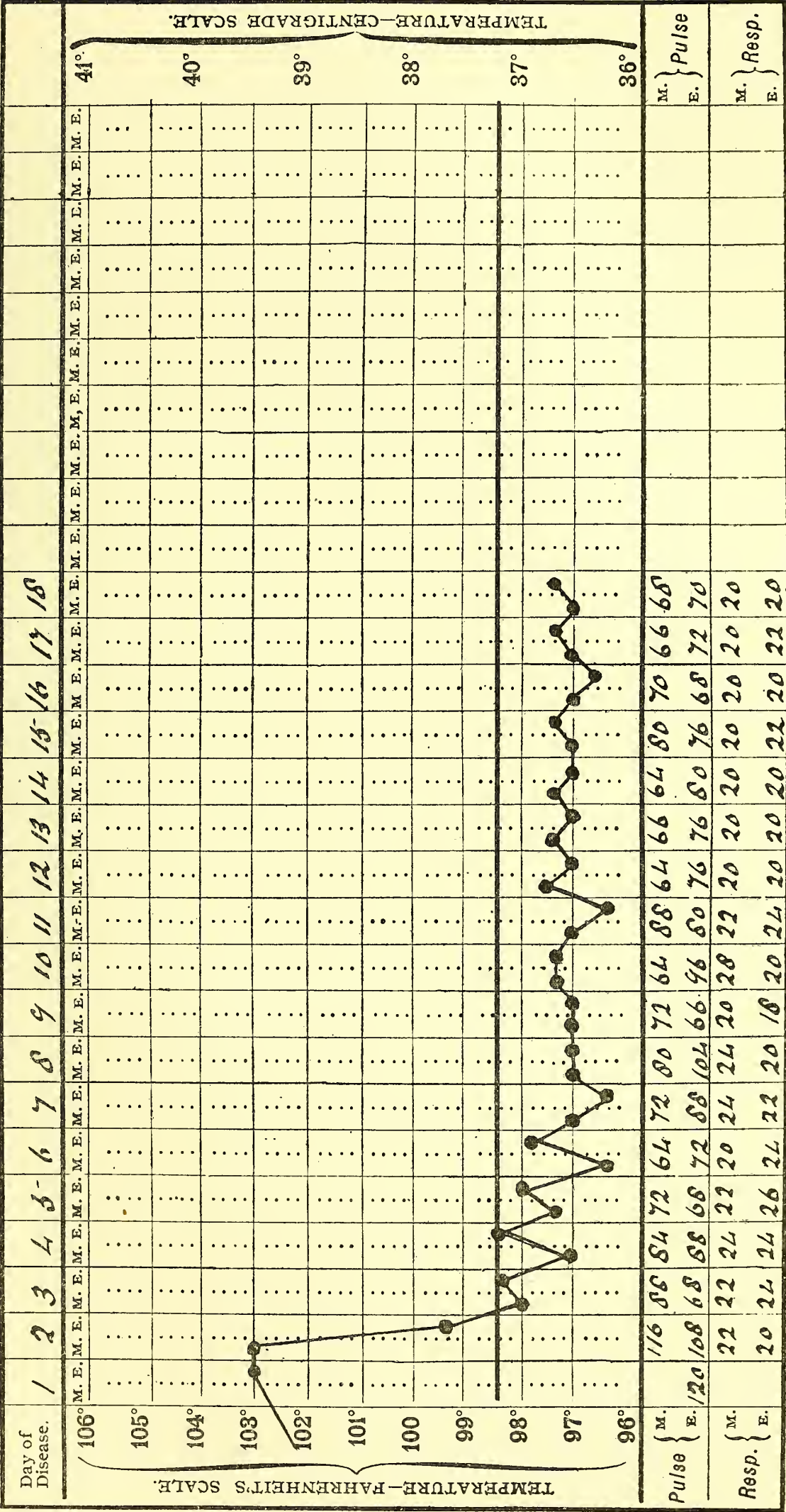


CHART 6.—Very Early Crisis (on the 2nd day) in Acute Pneumonia. Case of Staff-Sister Louisa C., aged 28 years, reported at page 436.



To this mild and transitory form of the disease the phrase *ephemeral pneumonia* also has been applied.

The following example of masked or larval pneumonia recently came under the writer's notice:—

Staff-Sister Louisa C., aged 28 years, on duty at the Meath Hospital, Dublin, fell out of sorts and suffered from a cold with shortness of breath during the early days of March, 1901. On Sunday, March 10, she became so poorly that she went to bed at 4 P.M. She complained of headache, malaise, shortness of breath, and a catch in her breathing. Next day she felt better and so continued until Wednesday, March 13, on the morning of which day she awoke with headache which got gradually worse until midday. At that hour (12 o'clock noon) she felt faint and had a severe rigor. The headache now became violent, and she suffered from increasing dyspnoea, so that she had to go to bed. Her temperature was now found to be 103° F. Next morning her aspect was typhus-like in the extreme—eyes suffused, pupils contracted, face dusky, eyelids heavy and drooping. P. 116, R. 22, T. 103°. Physical examination suggested a commencing right basic pneumonia. Towards evening the temperature fell to 99·4°, nor did it rise again, as the chart shows. The physical signs during the next few days were—a shade of dulness over the right base in succession to a remarkably clear percussion note; also increased vocal resonance, resembling pectoriloquy rather than bronchophony, and some crepitation (subcrepitus). The breathing was puerile over the left side. On Saturday, March 16, a patch of herpes labialis was observed near the left corner of the mouth.

In contrast to this—to quote Ernst Ziegler—it may happen that “during the course of an attack of pneumonia inflammation makes its appearance in other organs, such as the pericardium, meninges, and mediastinal tissue, the mucosa and submucosa of the pharynx, soft palate, and nasal cavities, the conjunctiva, kidneys, etc., in which case the affected structure can be shown to contain micrococci.” Such is complicated pneumonia—a condition which prolongs the fever movement, increases the risk to life, and may entirely upset the cyclic course of the disease.

In a paper on “Atypical Forms of Pneumonia,” Dr. E. Palier describes a case of what he calls “Gastric Pneumonia,” in which the symptoms closely resembled those of appendicitis. Abdominal section was performed by a consulting surgeon under the belief that disease of the appendix would be found. This, however, proved to be an error, and a post-mortem examination disclosed pneumonia associated with an empyema. Palier believes that abdominal symptoms in these cases of “gastric pneumonia” are to be ascribed to disturbance of the pneumo-gastric centre through the altered respiratory interchange. The abdominal pain is, in his opinion, analogous to the pain felt in the knee in hip-joint disease.

In March 1901, when epidemic pneumonia was rife in and near Dublin, the writer was called to see in consultation an anæmic girl, who for a week previously had been suffering from symptoms of acute gastritis, vomiting, pain, and tenderness in the left hypochondrium, and diarrhoea. At first her tongue had been thickly coated with a loose white fur, but at the time of the consultation this had been shed and the tongue was red and raw. A few hours before the patient had got out of bed, when a sudden faintness came on. This was followed by a rigor, and her temperature rose rapidly to 105·2° with a small thready pulse of 140. Physical examination revealed dulness over the base of the left lung, subcrepitus and tubular breathing intermixed, bronchophony, and, in front, some pleural friction. In three days she was dead, the fatal issue being brought about by exhaustion induced by incessant vomiting, tachycardia, and extreme feebleness of the heart's action. In this case one had to choose between an acute pneumonia with such severe gastric symptoms as to mask that disease in its early stages, or a perforating gastric ulcer leading to a left basic pleuropneumonia. Unfortunately a post-mortem examination could not be obtained, but in favour of the latter



diagnosis were the patient's age and sex, her anæmic state, and history of indigestion, and the sudden supervention of urgent gastric symptoms prior to the pneumonic rise of temperature and the development of the physical signs of an acute pleuropneumonia.

Had this patient been a child, a diagnosis of the "gastric pneumonia" described in 1880 by Adolf Baginsky, of Tübingen, might well have been made. In this form gastro-intestinal symptoms play a prominent part, while lung symptoms and signs do not obtrude themselves until just before crisis. Such cases may begin with vomiting, and diarrhœa is a conspicuous symptom.

At the extremes of life also acute pneumonia shows striking atypical peculiarities, which call for notice.

**PNEUMONIA IN CHILDHOOD.**—Apart from the fact that in very young children the exudation into the air vesicles is catarrhal rather than fibrinous, or croupous, in a large majority of cases, the clinical features of the disease are also often atypical. Gerhard, of Philadelphia, long ago pointed out that broncho-pneumonia, or catarrhal pneumonia, was the form of pulmonary inflammation which chiefly occurred in children under five years of age, whereas lobar or fibrinous pneumonia was much more common in children aged between five and fifteen years. Among 98 cases of catarrhal pneumonia, von Ziemssen reports 67 as having occurred before the third year; Steffen 52 in 72 cases during the same period. "All observations agree," writes Jürgensen, "that the first three years of life are those which are most often attacked by catarrhal pneumonia." According to Osler, lobar pneumonia has been met with in the new born, but he quotes Holt to show that of 270 cases of pneumonia in children under five years of age, 75 per cent were broncho-pneumonia. Notwithstanding this preponderance of a catarrhal exudation in the pneumonia of childhood, it is certain that the *Diplococcus pneumoniae* of Fränkel plays a part which is second to none in the ætiology of infantile pneumonia.

The atypical symptoms and course of pneumonic fever in childhood add greatly to the difficulty of diagnosis. "Even a physician of limited experience," wrote Jürgensen in 1875, "can easily recognise pneumonia in adults, but in *young children* this is by no means an easy matter. Everyone who has seen the mistakes of his assistants in the practice of a large polyclinic, will confirm this statement. Many children, who are reported to have died from 'teething,' 'worms,' 'convulsions,' etc., have really gone to their graves with an undiagnosed pneumonia."

Temperature usually runs especially high in patients under puberty—a fact which depends chiefly on the undeveloped mechanism of thermotaxis, —or body heat control—at tender ages, but also on the greater frequency of apical pneumonia in very young subjects. Probably the occasional appearance of an erythematous blush upon the skin, simulating scarlatina, is due to paresis of the vaso-motor nerves. When a rigor or more likely a convulsion, with vomiting, diarrhœa, redness of the fauces, and an erythema, usher in acute pneumonia in a child, a wrong diagnosis of scarlatina may be made for which there is some justification in this remarkable grouping of symptoms. Cerebral symptoms often mask pneumonia in childhood. The attack may be ushered in by convulsions. Osler speaks of "the so-called cerebral pneumonias of children," in which the disease sets in with a convulsion, and there are high fever, headache, delirium, great irritability, muscular tremor, and perhaps retraction of the head and neck. The diagnosis of meningitis is usually made, and the local affection in the lung may be overlooked. Again, from anatomical considerations, even fibrinous pneumonia is apt to assume a lobular character. So vascular is the pulmonary parenchyma in childhood, and so fine and delicate are the con-



nective tissue elements, that the spread of a pneumonia through contiguous lobules and even across the interlobar septa is an easy matter.

In children the adjacent serous membranes—pleura, pericardium, and endocardium—are very prone to infection by the *Diplococcus pneumoniae* and other pneumonic micro-organisms. In the case of a little boy, aged 3½ years, recently under the writer's care, the disease almost spent itself in producing a pericarditis with endocarditis. Only as an afterthought, as it were, did the physical signs of a left basic pneumonia show themselves.

A difficulty may arise in another way also from the circumstance that pleurisy with effusion may in very early life closely simulate pneumonia, not only in its fulminant clinical course with fever, cough, and dyspnoea, but also in its physical signs. Even in adult cases the blocking of a system of bronchial tubes by plastic exudation may interfere with the development of the classical signs of pneumonic consolidation—bronchophony, tubular breathing, and increased vocal fremitus. In such a case the dulness on percussion and absence of breath sounds—natural or adventitious—may be attributed to a pleural effusion rather than to a pulmonary consolidation.

Young children expectorate indeed, but do not *spit out*—the expectoration is commonly swallowed. This removes from our ken one easy means of diagnosis in pneumonic fever—the inspection of the rusty viscid sputum. But it does more than this by contributing to gastro-enteric derangement, the toxin-laden sputum when swallowed probably acting as a local irritant in the stomach and intestines. Hence vomiting and diarrhoea, tympanites, abdominal tenderness and pain, possibly convulsions or other cerebro-spinal symptoms.

Reference has already been made to the form of acute pneumonia in children to which Baginsky, of Tübingen, gave the name of “gastric pneumonia” in 1880.

When an attack of vomiting takes place, characteristic rusty sputa may be seen in the ejected matters, confirming the diagnosis of a croupous pneumonia, and proving that the expectorated phlegm has been swallowed in the first instance. As a diagnostic, E. Henoch lays stress on the character of the breathing, which is short, very rapid, with *grunting expiration*.

The pneumonia of childhood often runs a short course and terminates in recovery. Allusion has already been made to the remittent and relapsing character of the pyrexia in young children, a range of temperature which suggests a lobular distribution of the pulmonary trouble.

The danger to life from croupous pneumonia occurring in previously healthy children is trifling. Feeble children, however, succumb to the disease as easily as patients of more advanced age (Jürgensen). Henoch lost only 7 out of 64 cases in young children. Out of 201 cases in children under 16 years—the great majority being under 10 years—von Ziemssen lost only 7 (3·3 per cent). At the Kiel Polyclinic Jürgensen lost only 4 out of 110 patients under 10 years of age. Barthez lost only 2 out of 212 children aged from 2 to 15 years. In striking contrast to these figures is the heavy death toll which catarrhal pneumonia exacts from its subjects of tender age. Bartels lost all his patients under 1 year of age; after that period a little more than one-third. Von Ziemssen lost just one-half of the children under 1 year, about 38 per cent (17 out of 45) of those from 1 to 3 years old, only 25 per cent (8 out of 31) of those above 3 years of age.

*Pneumonia in Old Age.*—Insidious in its onset, and it may be latent in its course, pneumonia is a frequent and perilous disease of advanced life. By it in many a case the silver cord is loosed, when already strained almost



to breaking point by the presence of some other and older malady, such as carcinoma, system-diseases of the brain and spinal cord, chronic nephritis, gout, or other dyscrasia. The weak and degenerated heart of old age cannot battle against the added strain of a pneumonic fever and pulmonary consolidation, and so pneumonia is rightly dreaded by all physicians when it supervenes in the aged and infirm.

In its clinical features also the pneumonia of advanced life differs from the typical disease. Its seat is the most dependent parts of the lungs, whence perhaps it has some claim to the term "hypostatic pneumonia." It is less febrile, less acute, less lobar in its distribution. The sputum is less rusty, more cellular, than that of ordinary acute pneumonia, and so the attack works its treacherous way to the fatal end. During the "age of decline," as Jürgensen observes, pneumonia is one of the most dangerous diseases. The greater the wear and tear of the body (he adds) the more fatal the disease becomes. At the Greifswald Policlinic in ten years there were 941 cases of pneumonia with 102 deaths (= 10·8 per cent). From 40 to 50 years of age the mortality was 9·5 per cent, from 50 to 60 years, 20·0 per cent, from 60 to 70 years, 37·5 per cent.

If we inquire more closely how death is brought about in pneumonic fever we must agree with Jürgensen when he says: "It is the heart, and always the heart, upon which the burden is ultimately thrown;" and again: "The danger in croupous pneumonia threatens principally the heart of the patient. Death results from insufficiency of the heart." If this proposition be accepted as proved, we see why old people so readily succumb to this disease. The muscle of their heart is either wasted (simple atrophy), or it is fattily degenerated or infiltrated with fat, and so when the added strain of a continued fever and of a sudden extreme impediment in the lesser circulation is put upon it, the heart fails and death results.

Hence the necessity for daily, even hourly, observations on the heart's action and pulse during a pneumonia. The quality of the pulse as regards strength and volume and tension is of more importance than its mere frequency, and this applies to the cardiac impulse also. Undue rapidity of the heart and pulse-beat is unfavourable; still more so is any irregularity in volume or rhythm of the arterial throb, or an intermission in the beat. It is to be remembered that in acute pneumonia the normal ratio between the pulse and respirations is disturbed. That ratio in health is 4 to 1, perhaps more strictly 9 to 2. In pneumonia it may become only 3 to 1, or perhaps only 2 to 1. "In severe cases the frequency of the respiration approaches that of the pulse; the former may equal, and even exceed the latter." These are Jürgensen's words, and he adds: "Such cases, however, are very rare, but I have met with them, especially when the arteries were in a high degree atheromatous."

This disproportion between the pulse-rate and the respiration is diagnostic of pneumonia, for it fails to present itself in any other febrile disease except, perhaps, cerebro-spinal meningitis in its earlier stage.

#### PHYSICAL SIGNS

In 1896 Dr. Richard H. Kennan drew attention to the myotatic irritability of the pectoral muscles observed in acute croupous pneumonia, and expressed the opinion that the condition was a valuable early diagnostic sign of the disease—the more so as the myoidema is confined to the affected side. The writer has had many opportunities of confirming Dr. Kennan's observation, and can testify to the value of the



sign. To elicit the sign Dr. Kennan recommends that a finger of the left hand should be placed flat upon the chest in the direction of the course of the fibres of the pectoralis major. The pleximeter thus formed should be sharply percussed by the middle finger of the right hand, used as a plessor, when a rapid contraction immediately succeeding will be felt in the muscle under the finger of the left hand. Dr Kennan claims for this sign that it may precede the earliest inspiratory crepitations of pneumonia.

It will be convenient to analyse the physical signs which show themselves in the several stages through which the disease usually passes, namely—the stage of fluxion, or the preliminary stage described by William Stokes, the stage of engorgement or congestion (*engouement* of French writers), the stage of fibrinous exudation, or of red hepatisation or consolidation, and the stage of gray or yellow hepatisation or of resolution.

1. *Preliminary Stage of Stokes*.—The affected side shows lessened expansion and myotatic irritability. The percussion note is hyper-resonant, or tympanitic from relaxation of the lung tissue; or there may be a commencing dulness in patches. The breath sounds are harsh and rough, and there is already some increase in vocal resonance.

2. *Stage of Engorgement*.—There is lessened expansion. On palpation vocal fremitus is increased. The percussion note is abnormally clear, or even tympanitic in places, deficient in resonance in other places. The vesicular murmur is harsh, but at the same time weak. Towards the end of inspiration a fine crepitation is heard over the affected lobe. This is the *râle crepitant* of Laennec, to whom we owe so much of the nomenclature of diseases of the chest. Laennec compared this fine crepitus to the crackling of salt thrown upon a fire, but Dr. C. J. B. Williams made a more apt comparison when he likened it to the sound produced by rubbing one's own hair close to the ear between the fingers. Dr. Octavius Sturges imitated the crepitus of early pneumonia by the following manoeuvre: A fine sponge, dipped into a weak solution of gum, is lightly compressed within the hand; upon the gradual relaxation of the pressure a crepitus ensues which will be more or less fine, with the degree of dilution of the gum solution.

These comparisons are but of little value. The sound is one *sui generis*, once heard never to be forgotten—it is pneumonic crepitation.

3. *Red Hepatisation*.—In this stage the affected side may be slightly enlarged, especially in young patients, whose chest walls will more readily yield to pressure than the “rigid thorax” (Freund) of old age. Expansion is now markedly lessened. Tactile or vocal fremitus is equally increased, provided always that the bronchi communicating with the solidified part of the lung are not blocked, or that a pleural effusion is not already taking place. Dulness on percussion is now elicited over the solidified lung, but the note is not flat or absolutely dull, as in pleural effusion, but hollow, tubular, or perhaps slightly amphoric or metallic. Just before consolidation takes place the cracked-pot percussion sound (*bruit de pot fêlé* of Laennec) may be present.

Associated with dulness on percussion, a sensation of increased resistance is conveyed to the observer's hand when the chest wall is struck over the solid lung. The air-containing vesicles of the healthy lung rebound to the percussion blow, the blocked-up vesicles of the consolidated lung do not—hence the feeling of resistance, which is in proportion to the dulness on percussion.

Fine crepitation is still heard along the confines of the consolidated area, but for the most part the breath sounds now are bronchial or tubular—blowing, whiffing, or it may be metallic in character, unless the tubes are blocked. Vocal



resonance is increased, assuming one or other of the characters described as pectoriloquy (laryngeal or tracheal), bronchophony (bronchial), or ægophony (bleating bronchophony). The last-named variety indicates interference with the transmission of the vocal vibrations by the presence of fluid in the pleura. For two reasons the heart sounds are intensified over the area of solidified lung: first, the absence of the normal vesicular breathing throws them into relief; secondly, the solid lung acts as a conductor.

4. *Resolution*.—In this final stage the dulness on percussion may subside in patches, or generally, and in such a case with singular rapidity. At the same time a new crepitation is heard, particularly when the patient draws a deep breath. It is not so fine as the viscid crepitus of the earlier stage, and may be described as subcrepitant or medium—that is to say, the râles occupy an intermediate position between fine crepitus and coarse or mucous râle. This subcrepitus indicates the reopening of the air vesicles and the readmission of air, once the altered and emulsified exudation has been expectorated or absorbed. The expansile movements of the affected side of the chest gradually increase. At first they are voluntarily limited by the patient (vital ankylosis), because the freer movements put on the stretch any pleural adhesions which may have formed and so cause pain. Indeed, both after pleurisy and after pneumonia a temporary increase of that pain which is so well described as a “stitch in the side” is commonly observed as a result of the freer expansion of the chest wall.

#### COMPLICATIONS

“During the course of an attack of pneumonia,” writes Ernst Ziegler, “inflammation makes its appearance in other organs, such as the pericardium, meninges, and mediastinal tissue, the mucosa and sub-mucosa of the pharynx, soft palate and nasal cavities, the conjunctiva, kidneys, etc. in which case the affected structures can be shown to contain micrococci.” In this sentence we have primary infections by the *Diplococcus pneumoniae* indicated as a cause of complications. But there may be numerous secondary infections also by other pathogenic micro-organisms than those peculiar to pneumonia; and, in the third place, the marked leucocytosis in pneumonia, and other blood alterations, may give rise to complications. The white blood corpuscles increase from 10,000 to 19,000 or more per cubic millimetre; as many as 68,000 have been found. The increase is generally in the polymorpho-nuclear cells (Stengel). Fibrin is increased from 4 to 10 parts per 1000, so that a condition of marked hyperinosis exists.

One of the most frequent complications of acute pneumonia is *pleuritis*. In most cases the pleura is more or less involved in the morbid changes, so that the name *pleuropneumonia* is appropriate. In a comparatively small number of cases the affection of the pleura becomes so acute and widespread as to constitute danger to life. Then it becomes a complication. This happens in from 5 to 15 per cent of the cases of pneumonic fever. Usually the amount of effusion is small, but owing to the consolidation of the lung a comparatively scanty effusion may make a great show. The solid lung cannot collapse, and so the effused fluid passes upwards above the solidified lobe, which it also surrounds. The effusion may be fibro-serous, sero-fibrinous, or purulent (*empyema*). This complication is especially apt to occur in childhood, at which period of life the effusion is very commonly purulent from the outset.

In the aged, and also in young children, *bronchitis* plays a prominent part in the symptomatology of acute pneumonia, even when the exudation into the air vesicles is fibrinous or croupous. Just as there is in one



direction a certain amount of pleuritis in nearly all cases of pneumonic fever, so also there is almost always more or less bronchial catarrh in the other direction. This, however, does not count as a complication unless it is so widespread as of itself to cause risk to life through palsy of the bronchial muscle in the aged, or mechanically in children by blocking of the bronchioles by plugs of coagulated fibrinous exudation (plastic or croupous bronchitis). Acute capillary bronchitis, according to Magnus Huss, was present in 140 out of 2616 cases of croupous pneumonia observed in Stockholm—that is, in 5·4 per cent of the patients (Jürgensen).

*Pericarditis* and *endocarditis* are rare but perilous complications, particularly in cases in which the heart muscle is implicated, in myocarditis, or in hyaline or cloudy degeneration. Pericarditis may be the direct outcome of a bacterial infection, or may arise from an extension of inflammation from the neighbouring pleura to the pericardium by continuity. Endocarditis takes its origin in a bacterial infection, which may be either primary (diplococcic or pneumococcic) or secondary (streptococcic or staphylococcic). In the latter case the attack may assume the terrible characters of malignant or infective endocarditis, with early death. Andrew H. Smith says that pericarditis occurs in from 2 to 8 per cent of the cases of pneumonia. At the Presbyterian Hospital, New York, it was made out during life in 6 out of 485 cases. He shows that it is often overlooked during life, the area of the pneumonic dulness including that of the pericardial effusion, and auscultation not being practised at the time when the pericardial friction sound might have been heard. At Vienna there were 27 instances of pericarditis among 5738 pneumonia patients, or one-half (0·5) per cent.

*Collateral hyperæmia, gangrene of the lung, and abscess of the lung* must be mentioned as occasional complications intimately related to the primary lung affection. The first is brought about in part by the action of the right ventricle in driving the whole blood stream through the non-consolidated parts of the lungs. "The second factor," says Dr. Andrew Smith, "is the aspiration of an excess of blood into the sound lung by the action of the muscles of respiration. The inspiratory effort, not resulting in adequate expansion of the crippled lung, produces a condition of negative pressure in the sound one, and blood flows into the latter in consequence."

When the nutrient blood-vessels of the lung become blocked from thrombosis or by embolism, and saprophytic or putrefactive bacteria are present, the serious condition known as pulmonary gangrene results. Its occurrence is denoted by the stench from the breath and the horribly offensive sputa which the patient brings up, with much suffering in some cases. These sputa will be found on microscopic examination to contain strands of yellow elastic tissue—a proof that the lung structure is breaking up.

"In rare instances," writes Dr. Andrew Smith, "the affected portion of the lung undergoes a suppurative process. The surface has then a yellowish colour, and on section a purulent fluid exudes. The cells, resembling pus cells in all respects, not only fill the alveoli, but infiltrate the inter-alveolar walls. The pressure thus induced interferes with the nutrition of the septa, and may lead to their softening and breaking down. This may result in the formation of abscesses; but the condition, if at all extreme, usually proves fatal before this point is attained. Abscess of the lung, as an event of pneumonia, occurs in between 1 and 2 per cent of all cases. The abscess may have firm walls, or may be only an irregular cavity in broken-down tissue. Abscesses vary in size from that of a pea to the dimensions of an entire lobe. They sometimes discharge through a



bronchus; at other times, when small, they become encapsulated and undergo caseation." Traube first called attention to a change in the colour of the sputum to *green* as a diagnostic of suppuration in the lung.

*Jaundice* is an occasional complication of pneumonia. It occurred in 30 out of 5738 cases at Vienna (0·6 per cent), in 23 out of 2616 cases at Stockholm (0·9 per cent), but in 65 out of 230 cases at Basle (28·3 per cent), according to Fisser. Probably the last-mentioned high percentage included slight degrees of icterus, the result of hepatic congestion. Intense icterus is caused by a duodenal catarrh leading to obstruction of the bile ducts. Lastly and rarely, there may be a dangerous form of non-obstructive jaundice due to profound blood changes. G. Mante has shown that this variety depends on the hæmatolytic action of the *Diplococcus pneumoniae lanceolatus*.

*Parotitis* is a very rare but an exceedingly serious complication, which is met with in aged or debilitated patients. It is usually unilateral, according to Grisolle, and generally terminates in suppuration, or even in gangrene. In the reports of the Vienna hospitals this inflammation of the parotid gland is mentioned only six times in 5738 cases. Jürgensen never saw this complication—a sufficient proof of its rarity. Testi in 1889 obtained the *Diplococcus pneumoniae* in pure cultures from the pus in a case of parotitis complicating acute croupous pneumonia.

*Meningitis* may be caused by the *Diplococcus pneumoniae* directly, or—as pointed out by Jürgensen—the presence of croupous pneumonia is a powerful predisponent to epidemic cerebro-spinal meningitis. Of course, when Jürgensen wrote, the *Diplococcus intracellularis meningitidis* of Weichselbaum had not been isolated. The truth seems to be that the meningeal diplococcus undoubtedly sometimes gives rise to a pneumonia, while the pneumonic diplococcus with equal certainty may light up a meningitis.

Among other complications may be named *otitis media*, the purulent form of which may reinfect the lung; thrombosis of the femoral vein, usually the left; and various paralyses (including peripheral neuritis), which are, however, infrequent. *Diarrhœa* is also occasionally observed. The rarer sequelæ of pneumonia are: (1) Abscess of the lung; (2) Pulmonary gangrene, either circumscribed or diffuse; (3) Caseation, or "cheesy infiltration," in which a secondary tubercular infection often plays an important part; and (4) Fibrosis, or fibroid induration of the lung (chronic interstitial pneumonia, the "cirrhosis of the lung" of Sir Dominic Corrigan).

*Pulmonary consumption* very rarely follows in the wake of croupous pneumonia, whereas it is a common sequela of catarrhal pneumonia. In a draper's assistant, a young man aged 19, lately under the writer's care, a suspicious secondary fever set in after eight or nine days' pyrexia. Repeated examinations of the sputum failed to show the presence of tubercle bacilli, and the disturbance passed off after a fortnight, leaving no ill effects. The attack was probably due to some septic absorption in the resolution stage of a right apical pneumonia (see Temperature Chart I., page 429).

#### DIAGNOSIS

In a well-marked typical case the diagnosis of acute croupous pneumonia presents no difficulty, at least after a day or two. But in masked or larval cases, or where the disease runs an atypical and sluggish course, the diagnosis may be attended with much doubt. Again,



there are morbid states of the lung which in their symptoms and physical signs may simulate acute pneumonia.

*Typhus*.—The onset of pneumonia often closely resembles typhus in its pre-eruptive stage. The initial rigors, headache, sleeplessness, brisk rise of temperature, constipation, and suffused eyes and face, are common to both diseases. The diagnosis is settled by the respiratory symptoms of the lung attack, its rusty sputum, and its physical signs. Also the epidemic prevalence of either one or other disease should be taken into account.

*Enteric Fever*.—In the Annual Report of the Metropolitan Asylums Board for 1897, it is stated that of 293 patients admitted to hospital certified as suffering from enteric fever, 4 had bronchitis and 61 had lobar pneumonia. These figures would lead to the conclusion that enteric fever and acute pneumonia are often confounded with each other. The mistake might be excusable in those rare instances in which pneumonia runs a protracted course, with continued fever, and perhaps diarrhoea and wasting. But at the onset of an attack a systematic examination of the chest should obviate a wrong diagnosis.

Even when cough and expectoration are absent, the rapid respiration, unequal expansion of one side of the chest, and the results of a careful physical examination, should clear up the diagnosis, particularly if the Widal test yielded a negative result. Of course no reference is here made to the so-called typhoid or ataxic state, which may supervene in either enteric fever or pneumonia.

*Meningitis*.—Dr. Osler, writing in 1898, observes that “nervous symptoms are more frequent in pneumonia than in typhoid, and from the onset may so dominate that the local lesion is entirely overlooked. For instance, in the case of cerebral pneumonia of children, in which the disease sets in with a convulsion, there are high fever, delirium, great irritability, muscular tremor, and perhaps retraction of the head and neck, and consequently meningitis is usually diagnosed. Cases occur in which the malady sets in with acute mania. For example, a young man behaved so strangely on the train that he was handed over to the police as a lunatic, and as he had no cough and little fever (though he complained of a pain in the side) pneumonia was not recognised for several days. Again, pulmonary features are frequently masked where the patient has delirium tremens, and error is certain to occur unless it is made an invariable rule to examine the chest in these cases.” He goes on to say: “There are cases with toxic features resembling uræmia; without chill, cough, or pain in the side the patient may develop fever and a little shortness of breath, and then gradually grow dull and heavy, and within three days there may be a condition of profound toxæmia, with low, muttering delirium. In many of these cases the most characteristic symptoms of the disease may be absent, particularly the cough and the rusty sputum; but the physical signs, if they are elicitable, are well marked. Even in the gravest of these cerebral cases the crisis and the onset of convalescence may occur in the ordinary way, and the patient may pass from a condition of extreme danger to one of perfect safety.”

*Other Chest Affections*.—When in catarrhal pneumonia many adjacent lobules are affected, the physical signs of consolidation of the lung may be present. But the history of the attack should solve the question of diagnosis, although the distinction between catarrhal and croupous pneumonia may not matter much so far as treatment is concerned.

Passive or mechanical congestion of the lungs, leading to pulmonary oedema or hypostatic consolidation, may resemble pneumonia in regard to



physical signs. But the conditions named develop slowly, are for the most part afebrile, and are neither unilateral nor lobar in their distribution.

An apical pneumonia may excite fears of a pulmonary tuberculosis, which even the failure to discover Koch's bacillus in the sputum may not lull to rest. In a recent instance the writer was very uneasy about a patient, who when recovering from a sharp attack of right apical pneumonia developed a secondary fever of some ten days' duration, at the same time assuming a very phthisical aspect. Koch's bacilli were not found in the sputum, and the patient completely recovered. This secondary fever appeared to be due to absorption of some toxic agent in the exudation while undergoing resolution. The clinical chart in this case is given on page 428.

But it is with an acute *pleurisy* with effusion that pneumonia is most likely to be confounded, particularly in children. The onset of pleurisy may be sudden, with shivering, pain in the side, rapid and shallow breathing, with cough and fever. But the temperature does not run as high as in pneumonia, and the other evidences of a profound intoxication are wanting. The character of the cough is dry, short, sharp, and hacking in pleurisy, and the course of the attack is less cyclic than pneumonia. The sputa are indicative of catarrh or of pulmonary oedema in pleuritis.

Great attention must be paid to the physical signs, which in most cases afford grounds for a differential diagnosis. In the first place, on inspection, the natural furrows of the intercostal spaces are maintained in pneumonia, whereas in acute pleurisy with effusion they are effaced, the spaces sometimes bulging. On palpation, vocal or pectoral fremitus is usually exaggerated in pneumonia, lessened or lost in pleurisy. The cardiac impulse may be felt in or very near its usual situation in pneumonia; it may be widely displaced in pleurisy. On percussion the absolute dulness (amounting to flatness) of pleural effusion contrasts with the relative dulness of a pneumonic consolidation. And, lastly, on auscultation, we have in order in pneumonia rough breath sounds, fine crepitation, tubular breathing, and bronchophony or ægophony, with crepitus redux in the resolution stage. In pleuritis there are—in order—friction sounds, faintness or absence of breath sounds, or distant rhonchi and râles, with lessened vocal resonance. Signs of displacement of organs, namely, the heart, liver, spleen, and stomach, are present in cases of extensive pleural effusion.

Unfortunately, the principal signs may help but little in the case of young children, but in them pneumonia still generally keeps its cyclic and periodic character—the duration of the attack being numbered by days instead of weeks, as in pleurisy.

### PROGNOSIS

Pneumonia is always a serious disease, for its presence means interference with respiration and circulation alike. Yet the death-rate is not excessive in the United Kingdom.

It has already been shown that the mortality per cent of those attacked equals 14·5 in England and Wales. In the United States of America pneumonia appears to be a far deadlier disease than in the United Kingdom. According to Osler, pneumonia is the most widespread and the most fatal of all acute diseases. In the United States, during the census year 1890, 76,496 persons died of it, a death-rate per 100,000 of population of 186·94. "More deaths are attributed to it than to any single form of disease, except consumption" (Census Report). Hospital statistics, quoted



by Osler, show a very high rate of mortality—for example, Montreal General Hospital, 20·4 per cent; Johns Hopkins Hospital, 29·8 per cent; Pennsylvania Hospital, 29 per cent; Boston City Hospital, 29·1 per cent; and highest of all, in a southern climate—Charity Hospital, New Orleans, 38·01 per cent. Osler quotes Wells as collecting from various sources 223,730 cases, with 40,276 deaths, a mortality of 18·1 per cent.

The fatality of pneumonia is governed by the patient's age, sex, habits, previous state of health, the seat and extent of the lung affection, and the epidemic virulence of the disease for the time being.

*Age.*—Croupous pneumonia is not fatal to young children, or to previously healthy, sober adults. It is extremely deadly to old people, and in the presence of a broken-down constitution. "The greater the wear and tear of the body, the more fatal the disease becomes" (Jürgensen).

*Sex.*—Less common among females, pneumonia is more fatal to them than to males. Of 223 cases treated in the Presbyterian Hospital, New York, 170 were males, with a death-rate of 28·8 per cent; and 53 were females, with a death-rate of 31·2 per cent (Andrew H. Smith). One reason for the higher mortality among women is that the condition of pregnancy adds immensely to the danger of pneumonia, owing to the hyperinotic state of the blood which attends pregnancy.

*Habits.*—Alcoholic excess enormously adds to the peril of pneumonia. Among 428 cases admitted to the Presbyterian Hospital, New York, the mortality was 20 per cent among the non-alcoholic, 32 per cent among the moderately alcoholic, but 70 per cent among the markedly alcoholic (A. H. Smith).

*Previous Ill-Health.*—Damaged kidneys, of course, play an important rôle in increasing the mortality among alcoholic pneumonia patients. Rheumatism, malarial poisoning, diabetes, chronic nephritis, heart or lung disease, especially phthisis, all influence the prognosis unfavourably.

*Situation and Extent.*—An apical pneumonia is more dangerous than a basic attack, because it less readily resolves. Double pneumonia is very dangerous. So is an attack which involves the whole of one lung, or is accompanied by a croupous bronchitis (massive pneumonia). Right pneumonia is stated to be more fatal than left.

*Epidemic Influences.*—At times of epidemic prevalence the disease is apt to become virulent. The pneumonia of influenza is one of the deadliest of diseases, directly because it tends to spread through the lungs, and is accompanied by a general bronchial catarrh; indirectly, because of the concomitant heart-palsy.

Among unfavourable prognostics are high fever (above 105° F.), very rapid respirations without hyperpyrexia, tachycardia, and feeble pulse, accentuation of the pulmonary second sound followed by loss of this character (implying weakening of the right ventricle), absence of expectoration, or presence of prune juice expectoration, marked cerebral symptoms, gastro-enteric disturbance, with diarrhoea and tympanites, (in women) pregnancy. Excessive leucocytosis indicates suppuration. Moderate leucocytosis is regarded as favourable, and so are local eruptions of herpes.

## TREATMENT

In attempting to deal with this vast subject we propose to limit the scope of our inquiries by omitting the merely historical part of the question, and by boldly stating at the outset that the general principles of treatment of the infective fevers apply in their entirety to pneumonia fever also.



This disease, in a word, like them, is an essential or constitutional malady, self-limited in duration. No doubt its normal course is often interfered with and interrupted by accidental complications or sequelæ, which may themselves demand vigorous treatment. But to "cure pneumonia" is still, malgré the brothers Klemperer and their serum treatment, a feat as impossible of achievement as to cure typhus fever. "We know of no cure for fever; no man has ever cured it," wrote William Stokes, of Dublin, many years ago. "It is, however, curable spontaneously. If you leave it to its own course it is capable of curing itself. It will spontaneously subside. . . . We, so to speak, cure the patient by preventing him from dying. . . . Herein lies the secret of the treatment of fever. We watch the progress of the disease throughout its varying phases; we meet by judicious treatment, as they arise, the symptoms of secondary and local malady; we sustain the system as far as practicable; we preserve the sufferer at the least expense to the constitution; and we wait patiently until the hour shall strike when in accordance with the mysterious law of periodicity the fever shall have departed and convalescence shall have begun."

The "mysterious law of periodicity" is not now so mysterious as it was in the days of Stokes. The bacterial origin of the malady is everywhere admitted. The symptoms, local and constitutional, are due to a toxæmia. The toxin is resisted by the body-forces, as well as by an antitoxin which the toxæmia calls into being. Then, in the appropriate words of Dr. Andrew H. Smith, "the crisis is deferred until the infecting are balanced by the disinfecting forces, the latter including the various emunctories by which the poison is discharged from the body."

PROPHYLAXIS.—The prevention of pneumonia is a difficult matter. Perfect sanitation of the dwelling-house, civic cleanliness in the fullest sense, the maintenance of a high standard of personal health, and thorough disinfection or destruction of the sputum of pneumonic cases, which is dangerous only in a second degree to that of pulmonary consumption—such are the somewhat Utopian measures by adopting which we may hope to control pneumonia. The subjects of the malady should be isolated and treated in epidemic wards like any other fever patients. The researches of Netter (1888), Foà, and G. and F. Klemperer (1891) suggest the possibility of producing a temporary immunity against pneumonia even in man, by injections of an antipneumococcic serum prepared from an attenuated culture of the *Diplococcus pneumoniae*. The dog certainly can be rendered immune, and can also be cured of pneumonia by injections of anti-pneumococcic serum. Man, however, is relatively only slightly susceptible to the pneumonic diplococcus, and therefore we infer that he is equally but slightly susceptible to the antipneumococcic serum.

MANAGEMENT.—A patient suffering from pneumonia should take to bed at the earliest moment in a large, airy, warm, well-lighted room in a well-drained "fresh" house. Pure air and warmth and daylight are magical remedies in this as in other acute diseases. If the house in which the patient resides is badly drained he should at once be removed from its tainted air. Nothing is more striking than the marked improvement which often follows the removal of a pneumonia patient from insanitary surroundings to an airy, well-warmed, wholesome ward in a well-appointed general or private hospital.

Next to the place in which pneumonia is treated comes the manner in which it is nursed. As Graves said years ago, "It is of the utmost importance to economise the patient's strength in fever. The very act of lifting him up, or removing him from one side to another, tends to produce



exhaustion." Neither in pneumonia nor in typhus should the patient be allowed to assume the upright position lest cardiac failure should ensue. Hence the necessity for skilled nursing, which will save the sufferer from fatigue in a thousand and one ways. But the nurse must be one such as Shakespeare describes in *Cymbeline*—

"So kind, so duteous, diligent,  
So tender over his occasions, true  
So feat, so nurse-like."

The skilled nursing of pneumonia is then of paramount importance both by night and by day. It should be unceasing. One care the nurse should have is to prevent the patient talking—in pneumonia, speaking inflicts a far greater strain upon the patient's breathing than in other disease. It also induces fits of coughing, which exhaust the strength.

The dieting of the patient is all-important. As in enteric fever the food should be liquid, very nourishing, easily assimilable, given in moderate quantities at rather short intervals. More risk results to the patient from over-feeding than from starving. During the fever the total quantity of liquid food given in 24 hours should not exceed three pints and a half, that is, two litres. In addition cold water may be allowed *ad libitum*. Should the stomach be much upset and irritable, equal parts of whey and egg-water will usually be borne better than anything else. Egg-water—the *eau albumineuse* of the French—is prepared by whipping up the whites of from two to four eggs to a froth, then stirring into a pint of cold water and finally straining. This albumin-water replaces the casein of the milk which has been separated as curd in the making of the whey. Should a mild stimulant be required wine-whey may be substituted for ordinary two-milk whey—that is, whey made by adding one part of fresh butter-milk to two parts of warm milk in a saucepan over a slow fire. Milk and soda water, or carbonised water, form a grateful beverage, or the milk may be peptonised or given as "junket." Eggflip is a good food and stimulant combined. A light and palatable form is made by whipping up the yolk of a fresh egg, sprinkling a little powdered white sugar upon it, adding from a teaspoonful to a tablespoonful of whisky or brandy, and finally pumping soda water from a siphon upon the mixture in a tumbler.

As regards animal broths, it is to be remembered that beef tea as usually made is little more than a stimulant. It is better mixed with chicken broth. Strained mutton broth is very wholesome and palatable. But raw meat juice is much more nourishing than any of these, and mixed with port wine is a powerful restorative.

Freshly made tea with milk or cream may be allowed, and a little toast, if desired, may be soaked in the tea. A cup of black coffee, with or without brandy, is an excellent stimulant in the early morning after a restless night of tossing, sleeplessness, and delirium. Various fresh fruits, such as oranges, grapes, and bananas, may be given throughout the attack. Bananas, indeed, are very nourishing and digestible, and serve as food even in the presence of high fever.

The patient should not be allowed to suffer from thirst. Sipping cold water not only allays the thirst of fever better than anything else, but acts as a stimulant to both heart and liver, as pointed out by Sir Lauder Brunton. Sometimes thirst is relieved by simply holding a little cold or tepid water in the mouth and then spueing it out. Ice is often craved and should be allowed.

At this time of day it would be idle waste of time to give a detailed historical account of the various methods of treatment adopted in succession



for the "cure" of a periodic, self-limited disease. These methods were the antiphlogistic plan, by which it was sought to quench the fires of "*inflammation* of the lungs" by bleeding, calomel, antimony, and opium; the stimulant plan, so warmly advocated and energetically practised by Dr. Robert Todd, of London; the wise, if so-called expectant, method adopted by Dr. Hughes Bennet, of Edinburgh, in 1848, which though somewhat reactionary, recognised the principle of a "*vis medicatrix naturæ*" in relation to pneumonia; the antipyretic plan of the German school, with its large doses of quinine (30 grains at intervals of 48 hours), its repeated baths at 102° to 104° F., and its ice-pack or cold compresses to the chest walls; the symptomatic treatment of cough, pain, insomnia, delirium, vomiting, and diarrhœa, or other troublesome epiphenomena; and, lastly, the serum or antitoxin treatment, introduced by G. and F. Klemperer in 1891.

Our position to-day is this. We cull from one and all of the foregoing methods of treatment those measures and remedies which appear most likely to benefit our individual patients without slavishly following one method of treatment to the exclusion of the rest. Let us deal with the subject under two headings: (1) the Antitoxin treatment, (2) Rational treatment.

*Antitoxin Treatment.*—In their original communication on the nature of the infection in acute fibrinous pneumonia published in 1891, G. and F. Klemperer explain that the crisis seen in pneumonia in human beings occurs at the moment when the poisonous products, manufactured by the bacteria located in the lungs, are present in the circulation in amounts sufficient to call forth in the tissues the reactive change that results in the production of the antidotal substance which has the power of rendering the poisons inert.

At the time of the crisis in pneumonia the bacteria themselves are in no way affected. They remain in the lungs, and can be detected in full vigour and virulence in the sputum of patients a long time after the disease is cured. They have lost none of their power of producing poisonous products, and still possess their original pathogenic relations toward susceptible animals. It is only after the crisis that their poisons are neutralised by this antidotal proteid which has been eliminated by the cells of the tissues, and as this occurs the systemic manifestations gradually disappear. Klemperer and Klemperer isolated, from the cultures of the diplococcus of pneumonia, a proteid body which is the agent concerned in producing the tissue-changes that result in the formation of the protecting substance. They likewise isolated from the serum of immunified animals a proteid which possesses the same powers as the serum itself—of affording immunity and of curing the disease. The poisonous bacterial product they propose to call pneumotoxin; the protecting body, antipneumotoxin.

After obtaining these results upon the lower animals they directed their attention to human beings, and found that by the subcutaneous application of the serum of immunified animals to patients suffering from acute fibrinous pneumonia the results were in the main promising. They found that while healthy individuals and those suffering from other forms of disease presented no systemic reaction after the injection of the serum, in six cases of pneumonia in which the serum was employed there was a remarkable fall of temperature and slowing of the pulse within the first twelve hours after it was injected. In four of these cases the temperature fell to normal, but rose again after six hours. In two cases it fell to normal, and remained at that point.

Early in 1897 Dr. J. W. Washbourn, physician to the London Fever Hospital, reported two cases of pneumonia treated with antipneumococcic serum. The serum was derived from a pony which had been gradually immunised by injections into the subcutaneous tissue of the shoulder of broth cultivations of the *Diplococcus pneumoniae* heated to 60° C. for one hour, then of living agar cultivations, and, lastly, of living broth cultivations. In both cases the treatment appeared to exert a beneficial effect upon the disease. Washbourn recommends that the serum should be injected into the subcutaneous tissue with strict aseptic precautions. The dose should be 20 cubic centimetres (660 units). The treatment should be commenced as early as possible in the attack, and should be



repeated twice a day until the patient is convalescent. Andrew H. Smith thinks that we must conclude that up to the present the achievements of orrhoterapy in its application to pneumonia can scarcely be said to amount to more than an encouragement to further effort. "No really decisive results have been obtained. In some cases the effect seems to have been favourable, but in view of the variable course of pneumonia under all forms of treatment, it is impossible to assign to the injections any positive share in the result."

*Rational Treatment.*—Ever keeping before his mind the idea of the specific nature of pneumonic fever, the object of the physician in treating this disease should be to guide the patient safely back to health through the manifold dangers by which he is encompassed.

The bowels should be kept active once or twice a day. The diet should be regulated. Sleep should be secured.

Temperature may run so continuously high (104° F., or upwards) as to suggest a risk of heart failure, apt to be induced by the action of pyrexia, combined with the profound disturbance of the pulmonary circulation caused by the pneumonia. At the same time, we should recollect that the rate of mortality is higher in the presence of relatively low temperatures (under 103° F.) than it is when the thermometer ranges between 104° and 105°. The German doctrine of "*Das Heil-fieber*" holds good in the case of pneumonia as in that of other continued fevers. Elevation of body temperature means a reaction of the body forces against the toxæmia. Cold water, applied externally and taken internally, is the safest of all antipyretics. It lowers temperature without interfering with the production of body-heat (thermogenesis). Sir Lauder Brunton cites, as the most striking example he ever saw of the use of cold, the case of a patient suffering from pneumonia, who was dying from hyperpyrexia. At the suggestion of a Swedish physician who was present, Professor J. Hughes Bennet, of Edinburgh, under whose care the patient was, applied the wet pack. A big tub of cold water was ordered into the ward. All the bed-clothes were pulled off, a sheet was dipped in the water, and the patient was wrapped in it. In a few minutes it was taken off and a second cold sheet was applied. This was repeated again and again, and in an hour the patient lay quiet and comfortable, apparently in an easy slumber. From that time forward the patient went on without a bad symptom, and in due course recovered perfectly.

Sponging with vinegar and tepid water (one part in four) is a useful modification of the bathing treatment of pneumonia.

Another risk to life arises from collateral œdema of the lungs or brain, and this in a sthenic patient with high fever may be relieved by leeching or venesection, remedies which have been too much neglected of late years. Cyanosis, stupor, transitory paralysis affecting one or other side of the body (hemiplegia), rapid respirations, and a serous frothy expectoration, are indications of collateral hyperæmia, which a moderate venesection—4 to 6 or 8 ounces of blood—would probably at once relieve. The writer can call to mind three or four cases of this kind, in which venesection did good, and quickly.

The administration of alcoholic stimulants calls for much circumspection on the part of the physician. Many—probably most—pneumonic patients are better without stimulants, which may tire the heart and congest the liver. The intemperate and the aged require stimulants—the former in moderation, the latter often *pleno rivo*. It is a good plan to combine stimulants and food, as in egg-flip, wine-whey and egg-water, milk and whisky or brandy. For an emergency champagne plays a useful part. Women and children often bear stimulants badly. Under all circumstances,



the ordering of these dangerous remedies should be subject to revision day by day.

As secondary, though useful, aids to treatment, several local applications may be mentioned. A single leech applied early in the attack will often relieve the distressing "stitch in the side" of that stage. When the leech falls off the wound may be allowed to bleed into a linseed-meal poultice for a quarter of an hour or twenty minutes. A relay of hot linseed-meal poultices is an old-fashioned and useful practice. Mustard in varying proportions, when mixed with such a poultice, produces more decided counter-irritation, and prevents the uncomfortable feeling of chill which ensues on cooling of the poultice. In children a jacket of wadding is a good substitute for the poultice. The inflammable nature of wadding should not be forgotten. Turpentine stupes do good, particularly when there is coincident bronchitis. Dry cupping is excellent practice for the relief of pain and dyspnoea, but also as a preventive of collateral hyperæmia. Blistering is often harmful, and when prescribed should be used with moderation. Free and reckless blistering may damage the burdened kidneys, or actually set up a traumatic pleuropneumonia. Mention has already been made of the application of ice-cold cloths to the affected side, and even the ice-bag may be employed with advantage.

Two vital indications in treatment are—to support the heart and keep it going, and to prevent clotting of the blood.

The first indication is met by ordering digitalis or strophanthus, in full doses, singly or in combination with quinine, nux vomica, or strychnine. Digitalis is indicated when the pulse is rapid and of low tension, and when the kidneys do not act freely. This drug is well borne in pneumonia, and may be pushed. By some authors it is credited with an almost specific action on the disease. Strophanthus has the advantage of acting more quickly upon the heart, and may be given in high tension cases. Another useful drug is caffein, given as an effervescent citrate or hydrobromate, with or without 10-grain doses of salicylate of sodium. Caffein should be withheld late in the afternoon and towards night lest wakefulness should result. In threatened heart failure hypodermic injections of strychnine ( $\frac{1}{30}$ – $\frac{1}{15}$  grain) every eight or twelve hours often save life. Morphine may be given with it ( $\frac{1}{8}$ – $\frac{1}{6}$  grain) if the patient is restless and sleepless.

Blood-clotting may be anticipated by the exhibition of neutral salts, or of carbonate of ammonium in 5-grain doses every third, fourth, or sixth hour. A pill of 3 grains each of ammonium carbonate and quinine is a suitable form. Aromatic spirit of ammonium serves the same purpose; in urgent cases it may be combined with digitalis, Hoffmann's anodyne (compound spirit of ether), and tincture of bark. Inhalations of oxygen give great relief, and once tried are often craved for by the patient. The mouth-piece of the inhaler should not be taken into the mouth, for the concentrated stream of oxygen dries the tongue and parches the mouth. The writer has never seen any harm from the inhalation of diluted oxygen, the nozzle being held a couple of inches from and below the open mouth.

For the relief of cough the local applications already mentioned should be tried. Sipping cold water gives relief, or half an ounce of both glycerine and lemon juice may be put into a 2-ounce bottle, which should then be filled with water; of this simple mixture a teaspoonful may be sipped occasionally. To such a mixture cherry-laurel water, or syrup of codein, or solution of morphine, might be added to produce a more decided sedative effect. Sir William Jenner's cough mixture for phthisis should do good in some cases, but it is apt to check expectoration, namely:



R<sub>x</sub> Acidi hydrocyani diluti, ℥xlviij.  
 Liquoris morphinæ acetatis, ℥xcvj.  
 Syrupi aurantii, ad ʒiij.

*Signa*: A teaspoonful, by measure, to be sipped occasionally.

For the early headache and sleeplessness the phenazone and gelsemium draught, recommended in the article on "Influenza," may be prescribed. The formula for this magical remedy is as follows:—

R<sub>x</sub> Phenazoni, gr. xij.-xx.  
 Tincturæ gelsemii, ℥xx.  
 Aquæ chloroformi, ad ʒij.

*Signa*: One-fourth part every second or third hour until pain is eased.

As a rule narcotics, like opium or morphine, had best be avoided in the treatment of pneumonia. They lock up the secretions and check expectoration. Perhaps an exception may be made in favour of Dover's powder, which is useful when given in a pill with quinine and digitalis (Niemeyer's modification of Heim's pill).

The bromides are suitable hypnotics, exhibited with tincture of bark. If the heart is strong and early in the attack, chloral may be given in combination with the bromides of sodium, potassium, and ammonium; later on chloral is inadmissible except in young children, who bear the syrup well. A teaspoonful may be prescribed with 7 drachms of simple syrup or of aromatic syrup, the dose being one or two teaspoonfuls of this mixture at suitable intervals.

Chloralamide and bromide of potassium, of each 15 to 30 grains, sometimes act well in soothing and so inducing sleep. They may be dispensed flavoured with liquid extract of liquorice, or with tincture of orange peel and chloroform water.

Delirium is sometimes active in apical pneumonia, but it is seldom persistent. It is allayed by whatever makes the patient less restless and more comfortable. Sipping cold water for the relief of thirst, the application of cold compresses to the affected side, the relief of tympanites or flatulence or constipation, the application of warmth or cold to the forehead, the use of the menthol cone or of ice in stroking the brow—these simple means should in the first instance be tried. Only when they fail should more active measures be resorted to.

In coincident bronchial catarrh, or where "purulent infiltration" of the lung is taking place, turpentine is a sheet-anchor. It may be given in the form of capsules, or in punch or mixture. Turpentine punch is best made by rubbing a little lemon rind on a lump of white sugar, then dropping 15 to 20 minims of spirit of turpentine upon the sugar, and dissolving it in a wine-glassful of whisky punch.

Turpentine is prescribed in a mixture as follows:—

R<sub>x</sub> Spiritus terebinthinæ, ʒiij.  
 Ovi vitellum unum.

Tere et misce bene—

Spiritus ætheris nitrosi, ʒiij.  
 Spiritus chloroformi, ʒij.  
 Aquæ menthæ piperitæ, ad ʒviiij.

*Signa*: "Half an ounce to an ounce for a dose."

Other complications must, of course, be dealt with as they arise, but on all occasions and in all circumstances the physician should remember to avoid the "nimia diligentia medici" in treating a disease like pneumonia, which so pre-eminently is periodic, and tends to get well of itself, perhaps in spite of a fussy and ill-named "heroic" treatment.



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**Pneumothorax.** See PLEURA.

**Poisons.** See TOXICOLOGY.

Other references to poisons will be found in the following articles:—FOOD, ADULTERATION OF (vol. iii.); MORPHINOMANIA AND DRUG HABITS (vol. viii.), and TOXÆMIA.

**Poliomyelitis.** See PARALYSIS.

**Polyuria.** See DIABETES INSIPIDUS.

**Ponos** (πόνος, suffering, pain) is a peculiar endemic disease, affecting young children only, occurring in the Greek islands of Hydra and Spezia off the coast of Argolis. The disease is characterised by marasmus, enlargement of the spleen (usually), and a characteristic odour of the urine. The mortality is very great, either from the marasmus, hæmorrhage, or general collapse. Röser first directed attention to this disease in 1835, and Pallas called attention to the hæmorrhagic phenomena and their resemblance to those of scurvy in 1842, but it was not until 1871 that the disease was generally recognised by Grecian physicians. It is probably not so frequently met with now as it once was. Its origin is unknown. Jeanakopulos thought it was due to malarial infection, but this is not so. Climate and season have no influence upon its production, nor has the constitution



of the soil. It is met with in rich and poor alike, and food and water seem to have no effect in its production. But it seems to be limited to certain families, and is popularly supposed to be due to hereditary conditions, or at any rate to congenital predisposition, for the parents of children suffering from this disease are usually found to have suffered from some serious disease, especially tuberculosis. Karamitsas held that the disease was a kind of splenic leukæmia, but this view is not supported by either the symptoms during life or the post-mortem appearances. Hirsch describes it as a constitutional affection deeply rooted in errors of nutrition. Scheube considers that the disease much resembles the liver cirrhosis met with in India. In all probability some parasite will eventually be found to be its cause. The duration of the disease varies from two or three months to one or two years. It is rarely seen in children over four years of age, and Stephanos considers it rather more common in boys than in girls. Of the pathology of the disease we are quite ignorant.

The symptoms of ponos vary. The child suffers from fever of an intermittent or irregular type, fluctuations from first to last; if it assumes the character of hectic fever, death nearly always results; the child becomes feeble, pale, emaciated, disinclined to play, but the appetite is usually good; it may indeed become ravenous. *Pari passu* the spleen enlarges and is painful as a rule, and it is this pain which gives the name to the disease, but in some cases it is entirely absent, although the spleen is greatly enlarged. Constipation is the rule at first, followed later on by diarrhoea, sometimes severe. From the first and throughout the disease the urine has a peculiar characteristic penetrating odour, and bronchial catarrh usually obtains throughout the whole course of the disease. After the onset of diarrhoea œdema occurs, followed by ascites and sweating; and then hæmorrhage from the nose, bowels, and especially from the gums, occurs, together with ecchymoses of the skin. Pneumonia, meningitis, and peritonitis are apt to complicate matters, and lead to a speedily fatal termination of the disease.

*Treatment.*—This is unsatisfactory. If the child is attacked when very young a good wet nurse should be procured. Good food and tonics, such as iodide of iron, quinine, iron, are indicated; change of air should be advised, and on the bare chance that the disease may be possibly combined with a scorbutic element, plenty of ripe fruit should be given.

Dr. George I. Williamson, of Larnaca, Cyprus, writing of ponos, says:—“I have for some considerable time been on the outlook here for cases that might be so classified, but have never met with any. Many cases of malarial fever in infants seem to closely resemble the disease as described, say, in Manson's *Tropical Diseases*. From inquiries made, I find that ponos has never been even suspected to exist in Cyprus. Dr. Trepis, of Larnaca, a native of Hydra, writes me that the older medical men of that island considered ponos to be tubercle of the mesenteric glands and peritoneum, and that the inhabitants thought it contagious and infectious, as many children of the same family were frequently attacked; he adds that he does not know how the disease is now considered by the doctors of Greece, but his own idea is that it is not a disease *per se*.

I have lately come across an article on ponos written by Dr. Jiannakopoulos, who practised in Spetsee for a considerable number of years; his article appeared in a Greek medical journal in 1879, and so is of earlier date than the descriptions by Karahitsa (1880) and Stephanos (1881). Jiannakopoulos, whose article I have lately translated from the original Greek, considers the most suitable name to be septic marsh fever (ἐλογενής



πυρετὸς σηπεδονήδης), the title ponos (which he considers given by reason of the gangrene frequently noted) appearing unsuitable, as the terms πόνος and πονερά are used throughout Greece for any sore or wound. His idea as to ætiology is that a marsh miasm from the large marsh Berberonta is carried by the wind to Hydra and Spetsee, the sea being a good conductor of this miasm, which is, however, impeded by the presence of hills.

He mentions as symptoms the following—Beginning as an ordinary intermittent fever, it later becomes irregular: loss of strength and appetite, swelling of abdomen, characteristic septic odour of urine, straw colour of face and body; enlargement of spleen, but not of liver; necrosis of the jaws, especially the upper. Duration—months, not weeks.

Microscopically—no increase of leucocytes.

Boys and girls attacked equally often.

*Diagnosis.*—From dentition, leukæmia, malarial fever.

*Treatment.*—Quinine, good food, nursing, wine, cauterisation of gangrenous patches.

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**Pons.**—Lesions of the pons are of interest to the practitioner in at least two directions. Firstly, in cases where there is a gross lesion of the pons of a nature demonstrable to the naked eye, *e.g.* tumour growth; and in the second place there is a wider group of so-called nervous cases where some of the symptoms present are directly traceable to disturbance in this part of the central nervous system. For the due appreciation of either case we must have a clear conception of the physiology of the part, noting specially—

1. The part which the pons place as the transmitter of motor impulses from the brain cortex.

2. Its function as a transmitter of sensory impressions from the periphery.

3. The function of its grey matter as the nuclei of origin of the cranial nerves, especially the 5th, 6th, and 7th, and the close relationship of the 6th nucleus to the nucleus of the third nerve, whereby a lesion of the nucleus of the 6th nerve is attended by paresis or paralysis of that nerve, and also paresis of the internal rectus muscle on the other side (Conjugate Paralysis).

Bearing these in mind we will readily understand how a localised lesion in this region may produce very definite symptoms of a unilateral or bilateral nature; and if we bear in mind that the fibres of the facial nerve decussate in the lower half of the pons, at a level lower than the crossing of the pyramidal tract, we will understand how a condition of alternate or crossed paralysis (*vide* vol. ii., p. 60) may be brought about.

The diagnosis of a localised lesion in the pons is as a rule fairly easy, and is made from a study of the positive and negative symptoms present in each case (*See* art. on "Brain, Tumours of," vol. ii., p. 46). It is much



more difficult, and frequently impossible in cases of a more general nature, to determine how far the various motor or sensory disturbance is due to lesions in the pons rather than to disease in a region above or below that level. And, fortunately, in most cases the differential diagnosis is immaterial. The cause of the difficulty lies in the tendency for the occurrence of widespread vascular disease in these cases. Some diseases, for long regarded essentially lesions of the nervous system, are now looked upon as more of the nature of a general toxæmia with the chief local incidences in the nervous system, where the toxic agencies at work have exerted their influences primarily on the blood-vessels. In view of this, in all so-called nervous cases, more especially those of an ill-defined nature, it is well to carefully consider how far the anomalous symptoms may be due to vascular lesions in the pons or other part of the central or peripheral nervous system. This can only be determined by a careful study of the history and condition of each case, and in some cases the diagnosis of such vascular lesions can only be arrived at by a process of exclusion. The lesions of the pons met with in cases of cerebral hæmorrhage and in cases of Bulbar paralysis are referred to in their special sections (vol. ii., p. 4, and vol. ix., p. 144).

## Post-mortem Methods.

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THIS subject will be treated in two parts: the first describing the method of



performing a "post-mortem" and of recognising the more important diseased conditions which may be met with; the second, describing methods of preserving tissues and of preparing them for histological or bacteriological examination. By the former one can hope to demonstrate only gross, macroscopic lesions, but even with this in view the examination must be carried out in a careful, systematic manner. In many cases a definite diagnosis can only be arrived at after a further histological or bacteriological investigation.

## PART I.

### METHOD OF PERFORMING A POST-MORTEM EXAMINATION

RULES OF PROCEDURE, AND PRECAUTIONS TO BE OBSERVED.—*Personal*.—Before commencing examine the hands and wrists carefully for recent wounds.

Minute wounds which cannot otherwise be distinguished may be rendered manifest by the localised smarting produced by dipping the hands into weak solutions of ammonia or formaldehyde ("formalin").

If wounds exist, they must be sealed up with a solution of celloidin in equal parts of alcohol and ether. This is preferable to flexile collodion, as it adheres better to the skin. If this be not deemed sufficient protection thin rubber gauntlets, which must be absolutely water-tight, may be worn.

Anoint the hands and wrists with antiseptic vaseline, and wipe the palms so that the knife will not slip.

If the hands be cut during an operation, the wound should be encouraged to bleed by squeezing it under a stream of *cold* water, or by suction, then washed with a strong antiseptic and sealed with celloidin. If, soon after, such a wound become hot and tender, indicating infection, it should be opened up, painted with tinct. iodi. B.P., and a moist carbolic dressing applied. Immediate treatment often prevents grave consequences.

During the course of the operation keep the hands moist by an occasional dip into cold water, to which 1 per cent lysol, or 1:2000 corrosive sublimate may be added. Blood and discharges must never be allowed to dry on the skin.

On completion of the sectio, wash the hands thoroughly in *cold* water, preferably under a tap, using a nail-brush to remove all blood or discharges; secondly, in cold water with soap; then in hot water with soap. Finally, soak in 1:20 carbolic acid, 2 per cent lysol, or 1:1000 corrosive sublimate solutions, and wipe dry.

To deodorise the hands the best medium is a weak solution of "Sanitas" in water.

*Operative and Technical*.—In all post-mortem examinations system and cleanliness must be insisted on.

If possible, secure good light, preferably daylight.

In making incisions, always cut *away* from, never towards, the other hand, or the hands of an assistant, and in laying down a knife place it at some distance.

As a rule make long sweeping cuts with the belly of the knife, drawing it through, rather than pressing it into the tissues.

Use sharp knives. Blunt knives are dangerous, as more force is required, and this is apt to make the knife slip, with the attendant dangers.

If an organ is worth preserving make as few incisions into it as possible, consonant with the attainment of the desired information.



If a bacteriological examination be necessary, inoculations should be made at once into suitable media, or the organ conveyed whole to the laboratory, so that its investigation may begin as soon as possible.

#### INSTRUMENTS AND ACCESSORIES REQUIRED

Knives :—

One strong "cartilage" knife, for making the skin incision, and cutting through the costal cartilages.

One or more strong scalpels.

One curved, probe-pointed, bistoury.

One long amputation or "brain" knife for making sections of organs.

Bowel scissors.

Sharp pointed scissors.

In place of the two last, one pair of scissors may be used, with blades two to three inches long, one probe pointed, the other sharp.

A strong saw ; cutting bone-forceps ; strong dissecting forceps ; chisel and mallet.

Two strong needles curved in half their length.

Strong thin cord, or "Dutch" twine.

The above are essentials, and they or their equivalents are to be found in the armamentarium of every practitioner.

The following may be added :—

Lion forceps ; syringe with fine nozzle ; measure glass ; cones for measuring the diameters of orifices ; measuring tape or foot-rule ; balance and set of weights.

**METHOD OF PERFORMING A POST-MORTEM EXAMINATION.**—The following description applies to cases in which the whole body is to be examined. Frequently the examination is only partial, as necessitated by the requirements of diagnosis or the wishes of the relatives.

After laying out the instruments likely to be required, and, if in private, disposing sheets or newspapers so as to catch any blood or discharges, the first thing to be done, after noting the name, age, date of death and post-mortem, is to make an external examination.

#### EXTERNAL EXAMINATION

In special cases measure the length of the body and its girth at the shoulders. It is seldom necessary to weigh the body.

Note muscular development and state of nutrition.

*Rigor mortis.*—It is well at this stage to ascertain the presence and extent of rigor mortis, as subsequent movement of the body may abolish it in the neck and limbs.

Rigor mortis is a certain sign of death, and is caused by the coagulation of the myosin of the muscle fibres. Under ordinary circumstances it appears about five or six hours after death in the muscles of the head and neck, extending later to the trunk, then to the upper and lower extremities. When decomposition commences it passes off in the same order, from above downwards. Its onset and duration depend on several factors, such as temperature of the air, cause of death, and age of the individual.

In dry cold weather its duration is greater, the decomposition which abolishes it being delayed.

After exhausting illnesses, and those in which numerous convulsive muscular contractions have occurred, its onset is earlier than usual, and its duration short.

It begins earlier and passes off sooner in the young than in the well-developed adult.

It is often slight after death from sepsis, and passes off early.



It is practically never absent, though it may be almost imperceptible, for example, in puny, emaciated infants.

Examine the *surface* of the body from the head downwards to the feet.

Examine the *scalp* for cuts, bruises, and prominences, such as wens or tumour-growth. Note the condition of the *eyes* as to prominence; sub-conjunctival hæmorrhage, pointing to fracture of the bones forming the orbital cavity; inequality of the pupils.—These are usually of medium size, and equal.—Note the presence of eruptions on the face and around the mouth.

Note escape of blood or discharges from the nose, mouth, or ears. Bleeding from the ears is usually present in fracture of the base of the skull. If the blood come from the nose or mouth it may signify rupture of an aneurysm into the air passages or œsophagus; hæmorrhage into the stomach; hæmorrhage from the mucous membrane of the nose, nasopharynx, or mouth; or, fracture of the base of the skull.

Discharge of pus from the ear would draw attention to the condition of the middle ear and mastoid cells, and suggest the possibility of secondary intra-cranial suppuration.

Passing from the head to the *trunk* and *extremities*, one has to note the presence of wounds and scars, with their probable date, cedema of the surface, congenital marks and malformations, discolorations, disfigurations from accident or otherwise.

*Discolorations* may be of the following varieties: tattoo marks; pigmentation in connection with chronic ulcers, or the result of the occupation of the deceased; bruises and ecchymoses; hypostatic lividity; and discolorations due to decomposition. The last three are of importance.

*Ante-mortem bruising* is distinguished by a permanent discoloration which cannot be removed by pressure. If the part be incised the blood is seen to have escaped from the vessels, and to lie in the surrounding tissues.

*Post-mortem lividity* is a reddish or purplish discoloration occurring usually in dependent parts of the body, for example in the skin of the back when the body is lying with the face upwards. Over bony points the skin is compressed and lividity is absent, and it can be removed from other parts by pressure. It is caused by the blood-serum containing dissolved blood-pigment soaking out from the vessels into the surrounding tissues. As a general rule lividity is greatest in cases where the blood remains fluid, for example, in pronounced septic cases, or in cases of asphyxia due to accident or resultant on other diseases. In septicæmia and other morbid conditions in which there has been breaking down of the blood corpuscles setting free the pigment, the lividity may be general, and the superficial veins are distinctly mapped out, forming a close purplish network.

In ordinary cases, after twenty-four to thirty-six hours, signs of *decomposition* appear over the abdomen as a greenish discoloration of the skin. This is due to the action of sulphuretted hydrogen and ammonium sulphide upon iron compounds, black sulphides being produced in the subcutaneous tissues. This discoloration appears earlier over the site of suppurations in the tissues or cavities of the body.

The surface of the abdomen in women who have borne children, or in great distension from other causes, and that of the thighs and mammæ in fat women, may show numerous small silvery scars—"striæ gravidarum" or "lineæ albicantes"—due to stretching of the skin.

Enlarged glands in the neck, axilla, or groin may be felt readily through the skin.



In *rickets* in children the "rickety rosary," formed by thickening at the costo-chondral articulations, is readily made out, and at the same time the ends of the long bones are enlarged from the excessive proliferation of the cartilage cells at the epiphyses.

#### EXAMINATION OF THE BODY CAVITIES.

The Thorax and Abdomen are usually examined before the Head. To open them a median incision is made through the skin and superficial structures from the cricoid cartilage to the symphysis pubis, passing to one side of the umbilicus. The knife must be held with a full grip—never as one holds a pen—and the arm kept rigid, the movement being at the shoulder rather than at the wrist or elbow, and the cut being made with the belly of the knife.

After making this preliminary incision dissect down carefully in the epigastrium until on cutting through the peritoneum the liver comes into view; then, inserting two fingers of the left hand downwards into the abdomen along the line of the skin incision, draw the abdominal wall upwards and cut downwards between the fingers, opening up the cavity completely. If more room be required below, cut across the insertions of the recti abdominales from the peritoneal aspect.

Take hold in turn of the edges of this incision, and, pulling the flaps outwards, with bold sweeps of the knife reflect the whole of the superficial soft parts from the thoracic walls and costal margins as far as the nipple lines.

On first opening the peritoneum gas may escape from the cavity, indicating perforation of the intestine; escape of large quantities of fluid points to ascites.

Note *œdema* of the subcutaneous tissue, and any morbid change in the muscles—pallor, fatty or hyaline degeneration.

Note the position and relationships of the abdominal contents; the height of the *diaphragm*—normally on a level with the fourth right and the fifth left ribs; the relationship of the edge of the *liver* to the costal margin, crossing it normally in the nipple line at the level of the ninth right costal cartilage. This relationship may be altered by change in the size of the liver, or by alteration in the height of the diaphragm caused by increase or decrease in the volume of the thoracic or abdominal contents. Note any irregularity of the surface of the liver caused by cirrhosis, syphilitic cicatrices, or tumour-growth.

Passing downwards, note the extent of *stomach* wall visible, the amount normally visible not exceeding six to nine square inches. Note the position and appearance of the *colon* and *great omentum*, the latter being greatly thickened and shortened in certain cases of tubercular peritonitis, and showing a varying amount of fat even in health. Then, raising the omentum, along with the transverse colon, over the costal margins, look for general or partial distension or contraction of the *intestines*, and if present find out whether the condition have relation to a stricture or other obstruction—tumour or hernia—or be simply due to irregular peristalsis. Lymph, blood, pus, or fluid *fæces* may be present between the coils of intestine, the last indicating perforation of its walls. The coils of intestine near the gall-bladder usually show post-mortem staining with bile.

*Diverticula* may be found in connection with the small intestine, for example, *Meckel's*, connected to the ileum eighteen inches to three feet above the ileo-cæcal valve. *False diverticula* are simply the exaggerated pouchings of the large intestine met with in cases of *fæcal* loading.

*Peritonitis*.—The only indications of peritonitis may be general conges-



tion and dulling of the surface.—In *hypostasis* the posterior coils are congested, and the surface bright.—In peritonitis there is usually, however, a variable amount of lymph lying on the surface, or forming prismoidal collections between adjacent loops of intestine. The lymph may be simple, puriform, or may have organised, producing recent or old, general or localised, adhesions between coils of intestine, or between visceral and parietal peritoneum.

The older these adhesions are the less easily are they broken down. In general tubercular peritonitis they contain small or large caseous nodules.

Localised peritonitis may occur on the surfaces of the liver or spleen, round an appendicitis, or in the pelvis in the female.

As a result of old peritonitis bands of fibrous tissue may pass from one part of the peritoneal surface to another, forming loops in which internal herniæ may form.

*Intussusception* is a condition in which a portion of intestine is drawn downwards into another part, forming a firm, sausage-shaped mass which, if unrelieved, becomes gangrenous. The entering bowel becomes adherent by peritonitis to the orifice of the ensheathing bowel.

“*Agonal*” *intussusceptions* are occasionally found in children. They are usually multiple, only occur in the small intestines, and are distinguished by the absence of inflammation, and by the fact that the entering bowel can be readily disengaged.

*Volvulus* is a condition in which a sigmoid flexure with long meso-colon becomes twisted on itself, causing obstruction.

In *gangrene* of the intestine—for example, in strangulated hernia—the bowel becomes dark, livid, or greenish brown, and dull on the surface.

Note thickening, localised or diffuse, in the mesentery—the former due to enlarged glands, the latter to tuberculosis, sarcoma of glands, or mesenteric abscess.

Before removing the abdominal organs it is well at this stage to open the thorax in order to examine the serous cavities, and to make a general examination of the lungs and heart, particularly as to volume. If this be omitted the blood will drain away when the abdominal vessels are cut across, and the relationship of the thoracic organs will be disturbed.

For directions refer to the method of opening the thorax described later (see p. 471).

This being done, remove the abdominal viscera in the following order, which experience has shown to give the most satisfaction.

*Removal of Viscera.*—In the first place remove the *small intestine*. Taking up a coil, separate, with a sharp knife, the mesentery at its attachment to the bowel, and continue this until the whole small intestine is free. Then cut it across between double ligatures placed at the upper end of the jejunum and three inches above the ileo-cæcal valve.

It may be examined at once.

In miliary tuberculosis the serous surface presents numerous gray pin-head tubercles. These may be “general” throughout the peritoneum, or localised to the base of ulcers of the Peyer’s patches, which shine through the wall as dark purplish areas. They must not be confounded with miliary cancer nodules, which are usually larger, often bile-stained, and are usually surrounded by a narrow zone of congestion.

Next, placing the intestine in a sink or large basin, remove the ligatures and flush it with a stream of cold water, and with the scissors, the blunt blade being inserted into the bowel, cut it open along the line of attachment of the mesentery. The Peyer’s patches are thus uninjured, being placed opposite to that line.



Examine the mucous membrane for congestions and hæmorrhages, catarrhal enteritis, ulceration — typhoid, tubercular, or follicular — perforations, or for waxy degeneration in cases of chronic suppuration and phthisis pulmonalis.

In *catarrhal enteritis* the mucous membrane is congested, especially over the valvulæ conniventes, thickened and covered with tenacious mucus.

In *typhoid ulceration* the whole of the Peyer's patch is affected, hence the ulcer is usually longitudinal, with sharply cut, thin edges. The solitary glands are also affected. The floor of the ulcer is formed of the inner circular muscular coat.

In *tuberculosis* the ulcer in the Peyer's patch is irregular, with sinuous, worm-eaten outline and thickened edges. It tends to spread round the bowel like all other chronic ulcers. The floor is irregular and caseating; the mesentery is usually thickened and shortened. Miliary tubercles can always be made out under the serous surface.

In health little if any decomposition occurs in the small intestine, its functions being mainly digestive and absorbent. The fæces are fluid or semi-fluid.

The LARGE INTESTINE may now be removed. Placing the great omentum on the stretch detach it from the transverse colon. Then, commencing at the caput cæcum detach the colon and upper part of the rectum by cutting and tearing through their loose attachments. In removing the ascending part, be careful not to injure the kidney, or open into the duodenum to which it is closely applied.

Before dividing the rectum press its contents upwards, pass a ligature round its lower part and cut below the ligature. This procedure prevents soiling of the pelvis with fæces.

Wash out the large intestine, and cut it open along one of the longitudinal muscular bands. Open up the caput cæcum and slit up the *appendix vermiformis*.

The orifice of the appendix normally admits a probe; it may be constricted or obliterated. The cavity may be distended with mucus or contain foreign bodies or concretions resembling foreign bodies. Its walls may be ulcerated, gangrenous, or perforated. Its relations vary greatly under normal conditions.

*Catarrh*, with congestion of the ridges of the mucous membrane, is commonly seen in the large intestine. In infantile diarrhœa the intestine may present no morbid appearance. *Dysentery* is characterised by fibrinous deposit upon or in the mucous membrane, and sloughs may form and separate. *Tubercular ulceration* may occur somewhat similar to, but more extensive than in the small intestine.

*Syphilitic* ulcers are characterised by their serpiginous outline, undermined edges, and tendency to cicatrize. In *fæcal* accumulation—for example, behind a stricture—ulcers may be found in the colon.

*Tumours* of the large intestine may be non-malignant; for example, fibrous or myxomatous polypi and adenomata. They are more often malignant, tubular cancer being the most common, and occurring usually in the cæcum and at the flexures. The cancers proper are occasionally met with in the rectum, rarely elsewhere. Squamous epithelioma may occur at the anus.

*Mesentery*.—Examine for enlarged or tubercular glands. Apart from secondary metastases the only tumour to be looked for here is lymphosarcoma, commencing in the retro-peritoneal glands. Other tumours are rare.



The *Spleen* may now be removed. Its normal average weight in the adult is 6 oz. Its average length is from 5 to 6 inches; its breadth from 3 to 4 inches, and its thickness a little over one inch.

Its capsule may show adhesions or fibrous thickening.

In atrophy of the spleen the capsule becomes wrinkled and loose.

In chronic venous hyperæmia it is enlarged in all directions, firm and cuts crisply; its cut surface is flat, of a dark plum-colour, and shows numerous fibrous points and bands.

In acute hyperæmia the organ is usually enlarged, soft, and on section the pulp bulges, is soft and readily scraped away or washed away with a gentle stream of water. This is the "diffluent" spleen. It indicates pronounced septic infection.

In waxy change the Malpighian bodies may be specially affected—"sago" waxy—or the change may be "diffuse" throughout the pulp. These two varieties are usually combined to a greater or less extent. Application of a weak solution of iodine colours the waxy parts of a deep mahogany colour.

R $\bar{y}$ Iodine . . . . .	1 part.
Potass. iodide . . . . .	2 parts.
Water . . . . .	100 parts.

In general tuberculosis, miliary or caseous tubercles may be met with in the spleen.

The arteries of the spleen being of the nature of "end-arteries," blockage of them, for example by an embolus, results in the production of a "hæmorrhagic infarct," which, if the patient survive, becomes decolorised. This is usually wedge-shaped with its base to the surface, and projecting slightly upon it. Still later, the infarct becomes surrounded by contracting scar tissue, forming a groove on the surface.

Tumours are rarely met with in the spleen.

Gummata—tough necrotic areas of the consistence and colour of wet wash-leather are occasionally met with. The spleen may be greatly enlarged in leuco-cythaemia, ague, and occasionally in rickets.

Small accessory spleens or *spleniculi* are often present near the spleen, resemble it in structure, and share its morbid changes.

Remove the *liver*, *stomach*, *duodenum*, and *pancreas* in one mass. By so doing the continuity of the bile passages is conserved.

Cut through the suspensory ligament of the liver, freeing the organ from the diaphragm, then drawing it downwards, isolate and ligature the œsophagus as far up as possible. It is then easy to separate the organs above-mentioned from the posterior abdominal wall, care being taken not to injure the suprarenal capsules, particularly the right one, which lies close to the lower surface of the liver.

Placing the organs in their natural position, examine, firstly, the Stomach and Duodenum. Note the size of the stomach and presence of thickenings in its walls. It is dilated in stricture of the pylorus, small in stricture of the cardiac orifice or in general scirrhus of its walls. Remove the ligature and allow the contents to flow out into some receptacle.

These may be food, mucus, bile-stained material, clotted blood, or "coffee-grounds."

For investigation of poisons, etc., refer to article on "Toxicology."

Wash out the STOMACH AND DUODENUM, and slit them open, the former along its greater curvature, the latter along its convexity. If a stricture be present, preserve it intact.



Examine the *mucous membrane* of the stomach. Distinguish *post-mortem digestion* from *gastric ulceration*. The former occurs at the dependent part of the wall, hence usually at the cardiac end posteriorly. The walls become gradually thinned from digestion of the coats from within outwards, and if an opening exist in the centre of the thinned area, it has thin, frayed-out edges.

Gastric ulcers are rounded, "punched out"; if chronic they possess thickened edges, and are most commonly met with near the lesser curvature on the posterior wall towards the pylorus. Their base is formed by submucous or muscular coat, or by some subjacent organ, such as pancreas, which the ulceration has exposed.

*Hæmorrhage* may come from a gastric ulcer: from ulceration over varicose veins at the lower end of œsophagus—for example, in cirrhosis of the liver: from rupture of an aneurysm: from ulceration of a cancer: or the blood may have been swallowed.

In *acute gastric catarrh*, the mucous membrane is congested, swollen, œdematous, and covered with tenacious mucus. In *chronic gastric catarrh* the walls are thickened, the rugæ are prominent, and numerous rounded, mammillated, prominences may be present.

*Tumours*.—Papilloma at the cardiac orifice; tubular, hard and soft cancers at the orifices or in the body of the organ are met with. Tubular cancers are usually flat, the edges raised and slightly overhanging, their centres ulcerated. Hard cancers show great and firm thickening of the mucous and submucous coats, and hypertrophy of the muscular coats; they tend to contract. Soft cancers are characterised by their abrupt, thick, rampart-like edges, their fungation, soft consistence, and great tendency to ulcerate in the centre. Carcinoma of the stomach may undergo colloid degeneration. Squamous epithelioma may spread from the œsophagus to the cardiac end of the stomach.

*Duodenum*.—*Ulcers* similar to those in the stomach are not unfrequently met with in the first part.

*Catarrhal inflammation* shows characters similar to catarrh of the rest of the alimentary tract.

*Stricture* may occur at the entrance of the biliary and pancreatic ducts; and tumour growth, non-malignant or malignant, may also occur there, the latter often as an extension from the head of the pancreas.

Lay the liver on its upper surface and arrange the duodenum so that no kinking of the common bile-duct can take place, and by pressure on the gall-bladder endeavour to force bile downwards into the duodenum. If it do not pass, lay open the bile-ducts carefully throughout their whole length, and examine for stricture, compression by scar tissue or tumours, or for the presence of calculi blocking them.

Next lay bare the *pancreas* from the front; note its consistence, and any pronounced enlargement, then slice it longitudinally to determine whether any tumour-growth be present, and transversely to determine the amount of fibrous tissue present. Note whether it be softened and hæmorrhagic (hæmorrhagic pancreatitis) or show suppuration or gangrene. It is not necessary in ordinary cases to expose its duct. Occasionally the pancreas is found ruptured in cases of abdominal injuries. The pancreas weighs from 3 to 4 ounces in the adult.

The gastro-hepatic omentum may now be divided and the liver separated and examined.

THE LIVER.—The normal average weight of the liver is 3 lb 5 oz. in



the male, and 2 lb 12 oz. in the female. It measures transversely from 10 to 12 inches, antero-posteriorly from 6 to 8 inches, and its greatest thickness is from 3 to 5 inches. Relatively it is larger in the child than in the adult.

It is of a chocolate colour, and its substance can be distinguished through the capsule, which is thin, smooth, and glistening.

The lobules, which vary in size from  $\frac{1}{16}$  to  $\frac{1}{20}$  of an inch, can be faintly distinguished. On the cut surface the openings of numerous large veins are seen. When laid on a plane surface the organ flattens out.

*Morbid Conditions.*—The capsule may show localised or general fibrous thickening. The surface may show transverse or antero-posterior grooves, the result of compression, for example by corsets. The organ may be displaced from the same cause. It may be ruptured from injury.

*Degenerations and Infiltrations* of the liver.

*Cloudy Swelling.*—This usually occurs in death with high temperature. The organ is paler than normal, the capsule tense, the cut surface smooth and glistening, with the individual lobules somewhat enlarged and outlined by a rather indefinite pale zone.

*Fatty Infiltration.*—The organ is enlarged, brown or orange in colour, the consistence lessened, the individual lobule much increased in size, and outlined by a distinct pale or yellow zone. In fatty hypertrophic cirrhosis the organ is greatly enlarged, and may be of a canary yellow colour throughout.

*Waxy or Amyloid Disease.*—The organ is greatly enlarged, its consistence is firm, and it preserves its shape when removed from the body. It is pale, translucent, and of a pinkish gray colour. The lobules may be outlined by a yellow zone of fatty infiltration. The edge of a section remains sharp. The lobule is greatly increased in size, measuring  $\frac{1}{16}$  to  $\frac{1}{8}$  of an inch, and the intermediate zone is marked out as a distinct, grayish, translucent ring. Application of the iodine test (*vid.* spleen) renders the waxy change very evident.

*Fatty Degeneration.*—The organ is smaller than normal, the capsule apparently thickened, and is wrinkled. The organ is soft, very friable, of a dull orange colour; the lobule may show a ring or spot of a yellower colour, according as the central or peripheral zone is affected. Either zone may show the change, the former quite as often as the latter.

In *chronic venous hyperæmia* the central zone of the lobule is marked out as a dark brown spot. This gives a mottled appearance, “nutmeg liver,” to the organ. It is much enlarged and may be firmer than normal. The condition results from chronic valvular disease of the heart.

*Multilobular Cirrhosis.*—In this condition the surface of the liver is covered with rounded projections, varying in diameter about from one-twentieth of an inch to about one-fifth of an inch. Separating them is a network of shallow grooves, caused by indrawing of the capsule by scar tissue formed along the portal vessels. On section a similar network is seen of pinkish gray colour separating opaque orange coloured islets of liver tissue. The organ is usually diminished in size, but may not weigh less than normal. The condition gives rise to ascites and chronic venous hyperæmia in the portal venous system.

*Monolobular Cirrhosis.*—The organ is usually increased in size, the capsule smooth or only slightly wrinkled, and the newly-formed fibrous tissue is made out with difficulty, as the network it forms is finer, enveloping individual lobules.

In *acquired syphilis gummata* may form in the liver. *Congenital syphilis* is associated with a diffuse fibrosis of the liver.

In *acute yellow atrophy* the organ is much lessened in size and flabby, the capsule being wrinkled. On section it shows alternating yellow and rusty red patches.

*Suppuration.*—*Tropical* abscesses are usually single and of large size, and the liver substance shows degenerative changes.

Abscesses from other causes are usually small and multiple; they may be part of a general *pyæmia*, of a pyæmia confined to the portal system, or may result from a suppurative inflammation of the bile-ducts (*cholangitis*).

*Tuberculosis.*—*Miliary* tubercles occur as minute gray points best seen under the capsule. *Caseous* tubercle occurs as small yellow points or larger bile-stained softening areas.

*Parasites.*—The most common parasite is the cystic stage of the *Tænia echinococcus*—the “hydatid cyst.”



The cyst wall is formed of thin laminae, of a material resembling the boiled white of a duck's egg. It is smooth and glistening on its outer surface, but granular on its inner surface. It usually contains secondary cysts. In one variety the secondary cysts form in the tissue round the primary cyst, producing the "multilocular" hydatid.

The important *new-growths* are malignant, and may be primary or secondary. The former may be single, the latter are practically always multiple. They may be of the nature of cancers—hard, soft, or tubular, non-pigmented or pigmented, or they may be sarcomata.

In *hard* (scirrhus) cancer the nodules are usually of firm consistence, and have a sharp outline. They tend to retract rather than degenerate towards their centre, so that an appearance of hollowing or "umbilication" may be produced on the surface. This is, however, by no means confined to the hard cancers.

In the *soft* cancers (medullary or encephaloid) the tumour is usually rounded, soft, and spongy. It tends to degenerate early in its centre.

In *tubular* cancer (malignant adenoma) the nodules are usually numerous, rather irregular in outline, on the surface showing more variegation of colour than the others, for example, pink, yellow, and creamy areas. In this variety, especially, the liver is greatly increased in size.

The *sarcomata* are usually secondary, may be cerebriform in appearance and consistence—round-celled sarcoma—or firmer and fasciculated—spindle-celled sarcoma.

The *pigmented* (melanotic) tumours are necessarily secondary, and are readily distinguished by their sepia coloration. If the primary tumour be discernible, this will settle their epithelial or connective tissue origin. Usually, however, microscopic examination is necessary.

### Resumé

The liver is *diminished* in size in—

Fatty degeneration, simple atrophy, acute yellow atrophy, late multilobular cirrhosis, and often in cancer of the pyloric end of the stomach.

It is *increased* in size in—

Cloudy swelling (slightly); fatty infiltration, unilobular cirrhosis especially with fatty change; waxy disease; chronic venous congestion; cancer; occasionally in early multilobular cirrhosis and in obstruction of common bile-duct. It is also enlarged, but irregularly, in abscess formation, and in presence of hydatid cysts.

The substance is *paler* than normal in—

Cloudy swelling; fatty changes; waxy disease; cirrhosis.

It is *darker* than normal in—

Chronic venous congestion; in pigmentary conditions and tumours.

Its consistence is *softer* in—

Fatty changes; acute yellow atrophy.

Its consistence is *firmer* in—

Cirrhosis; waxy disease; late stages of chronic venous congestion; fibrosis from syphilis or other cause.

The only organs now left in the abdomen are the *kidneys* and *suprarenal capsules*.

The latter are not usually examined closely, unless for some reason or other attention is specially directed to them.

They may show *waxy* degeneration along with other organs, where chronic suppurations or tertiary syphilitic conditions exist. They are the seat of tubercular *caseation* in Addison's disease, and occasionally show tumour-growth, usually secondary. In cases of toxæmia the medulla may become so soft that, in removal from the body, it breaks down, and the organ comes to resemble a cyst bounded by the more resistant cortex.

The *Kidneys* must always be carefully examined.



To remove a kidney, cut through the peritoneum at its outer margin and peel it off the surface, then raise the organ out of its bed, separating the structures surrounding it until it is attached only by its vessels and ureter. Cut these across at some distance from the hilum, noting whether any fluid escapes from the ureter, and if so, its character, whether clear or turbid. If the ureter or ureters be dilated, it is advisable to remove the whole urinary tract, including part, at any rate, of the corpus spongiosum urethræ, particularly if stricture of the urethra be suspected.

After examining the surface and noting abnormal firmness of perinephric fat, presence of perinephric abscess, or congestion of the surface, cut it open from the outer margin inwards towards the hilum, being careful not to injure the hand holding it. Then examine in order—

Capsule.

Cortex.

Medulla.

Renal pelvis and ureter.

In normal conditions in the male the left kidney weighs, on an average,  $5\frac{1}{2}$  ounces, the right kidney  $5\frac{1}{4}$  ounces, in the female being about half an ounce lighter. The left is usually longer and narrower than the right, but this distinction does not always hold, and one has to determine the side of the excised kidney by the position of the structures entering and leaving its hilum; the vein is in front, the artery in the middle, and the ureter behind and at its lower part.

If fat surround it, it should separate easily. The capsule is thin, translucent, and when a section of the organ is made should strip readily. On the cut surface the cortex should bear to the medulla a relationship of about one to three, and should be of a dull, brownish colour, but paler than the medulla.

In children a lobated arrangement is apparent up to about the third year, and on section the interpyramidal cortex may also be separated into two, each part in connection with its own pyramid. This condition may persist—"foetal lobulation."

The two kidneys may be connected at their lower part by kidney substance or by a fibrous band. This is the "horse-shoe kidney." The ureters always pass in front of the connecting bridge. The malformation does not interfere with function.

If one kidney become functionless the other undergoes compensatory hypertrophy.

A uniform cystic transformation exists in the congenital cystic kidney.

If a sharply circumscribed area of an otherwise functioning kidney be cystic, it usually indicates an adenomatous new-growth.

If scattered cysts exist in a firm, functioning kidney, they are retention cysts, due to disease (chronic interstitial inflammation) of the organ, or to other causes obstructing the tubules.

*General Characters of Disease in the Kidneys.*—A soft, flabby kidney indicates parenchymatous change.

A kidney of firm consistence indicates an interstitial change.

A *very* easily stripped capsule indicates a parenchymatous change.

A thickened and adherent capsule indicates interstitial change of some standing. In removing it, portions of kidney substance adhere to it.

A thickened subcapsular cortex usually indicates parenchymatous change, particularly if pale, or patchy, or yellow.

A thickened inter-pyramidal cortex is usually present in interstitial inflammation.



An atrophied cortex, associated with adherent capsule, indicates chronic interstitial inflammation.

Marked increase of fat round the pelvis indicates atrophic changes in the kidney such as occur in chronic interstitial nephritis, or arterio-sclerotic atrophy.

Yellow spots in the kidney substance, surrounded by a zone of congestion, are suppurative in origin.

Gray or yellow spots, without a zone of congestion, are tuberculous in origin.

Distinct dilatation of the pelvis of the kidney—*hydronephrosis*—occurs only from obstruction below in the urinary passages. Unilateral hydronephrosis results from obstruction or stricture of the ureter corresponding. Bilateral hydronephrosis occurs most commonly in stricture of the urethra or phimosis.

Hydronephrosis is to be distinguished from *scrofulous kidney*. In this large areas of the kidney have caseated and broken down into the pelvis, so the remains of the kidney bound an irregular cavity with diverticula, as in hydronephrosis. Here, however, the cavities are rough-walled, lined with caseous material, whereas in hydronephrosis the walls are smooth, lined with mucous membrane. The two conditions may be conjoined.

*Waxy or Amyloid Disease*.—Here the history of the case helps greatly to a diagnosis.

The waxy change occurs along with other parenchymatous or interstitial changes in old chronic suppurations and excavations of pulmonary tuberculosis, and in tertiary syphilis. The organ is enlarged, sometimes very greatly, as when the condition accompanies parenchymatous inflammation. It is soft, pale, of a light, pinkish gray colour, with streaks and patches of yellow in the cortex. The tips of the pyramids are pale, the boundary layer still remaining the darkest part of the organ. In the cortex the Malpighian bodies stand out distinctly as translucent gray points, and the iodine solution displays well the waxy glomeruli and straight vessels.

When there is accompanying or pre-existent interstitial change the organ is slightly enlarged, firmer than normal, its general appearance on section resembles that of the other variety, but the patchy pallor exists throughout the organ, the boundary layer being slightly, if at all, redder than the rest of the surface.

*Infarcts of Kidney*.—These are localised areas of necrosis, simple or suppurative, caused by embolic blocking of an interlobular artery. The area corresponds to the portion supplied by the artery. They are pyramidal in shape, the base towards the capsule, and slightly raised above its general surface. They are pale and surrounded by a hæmorrhagic zone. Organised infarcts are seen as scars, causing a localised adhesion and indrawing of the capsule.

*Calculi*, irregular and brownish, may occur in the pelvis of the kidney or in the ureter at its lower end, where it enters the bladder. This is the narrowest part of the tube.

With the indications above mentioned the more common morbid conditions of the kidney and ureter can be, at all events approximately, diagnosed with the unaided eye.

**ABDOMINAL PELVIS**.—In peritonitis lymph or pus may be more evident in the pelvis than elsewhere in the abdomen, having gravitated there.

*Male Pelvis*.—Examine the pouch of Douglas for effusions, pus, or fluid fæces which have escaped from a perforation of the bowel.

*Bladder*.—Note the state of the bladder, distended or empty. Try to express its contents through the urethra, to show whether this be patent or strictured. The bladder may be catheterised or opened *in situ*, and the condition of the urine and inner surface of the bladder ascertained. If the urine be decomposing there is probably cystitis.

Cystitis is indicated by congestion of the surface and rugæ, which may be covered with tenacious mucus.

Examine the walls for hypertrophy. In this condition the muscular rugæ are very prominent. Do not confound this with the apparently thickened and rugose walls of the contracted bladder.

Remove the bladder, separating the urethra from its connections to the arch of the pubis, and cutting it across at the posterior part of the bulbous portion. Slit up the bladder, prostate, and urethra along the



anterior surface. Examine the prostate for hypertrophy, tumour-growth, suppuration, tuberculosis, or calculi.

In cases which demand their examination expose the *vesiculæ seminales* on the outer posterior wall, and make sections. They present a honey-combed appearance, and contain normally a clear, viscid, slightly yellow fluid. They may be thickened and full of pus, or may show tuberculous change.

The *rectum* is usually removed along with the bladder, and should be slit up along its posterior wall. It may show congestion and catarrh, or tumour growth—polypi (adenomatous), the cancers proper, or squamous epithelioma. Occasionally non-malignant stricture is met with, for example, after syphilitic ulceration (in women).

Remember that in the rectum normally there are two projecting semi-lunar folds of mucous membrane, which must not be mistaken for new formations.

*Female Pelvis.*—Note adhesions from old peritonitis. These are often seen crossing the pouch of Douglas.

In all cases in women in which malignant tumours occur higher up in the abdomen, particularly in the liver, look for possible primary growth in the uterus or ovaries.

The body of the uterus in the adult projects about 3 inches behind and  $2\frac{1}{2}$  inches in front upwards into the pelvis, the peritoneum thus covering it completely on its posterior surface, and in great part on its anterior surface. It is about 2 inches broad at its fundus and about an inch thick. The cervix uteri is not visible from the pelvis.

The uterus may be enlarged and project into the abdomen in pregnancy and in internal tumour-growth. Generally speaking, these conditions may be distinguished by the position of the Fallopian tubes which leave the uterus high up in the former case, and low down in the latter case. Fibroids of the uterus usually cause irregular enlargement of the body of the uterus, and if sub-serous, may form large projections from its surface.

In advanced malignant new-growths originating in the cervix, the lower part of the uterus may be destroyed, leaving an irregular cavity bridged across transversely by the remains of the fundus and the Fallopian tubes attached to it.

The *Fallopian tubes* pass outwards from the cornua of the uterus along the upper borders of the broad ligament. They vary in length, and their outer extremity, which is fimbriated, turns more or less backwards. The outer end may be occluded, leading to dilatation of the tube with watery, purulent, or hæmorrhagic fluid. Enlargement of the tube may be due to salpingitis, tubal gestation, or tumour-growth. The internal lining membrane is normally thrown into numerous longitudinal folds, which are most evident towards the outer extremity. A small pear-shaped, thin-walled cyst, the hydatid of Morgagni, is frequently seen attached to the fimbriated end, and is not abnormal.

The *ovaries* are attached by a fold of peritoneum to the posterior surface of the broad ligament. They are usually about the size of an almond in its shell. In old age they become hard, atrophied, and scarred on the surface.

In *peritonitis* they may be covered with lymph, which may be purulent. If inflamed, they are enlarged, soft, and congested, and may contain abscesses.

*New-growths* may be solid—cancer, sarcoma, fibromyoma—or cystic.



The cystic growths are important. They may be of great size and weight, projecting far up into the abdomen.

They may be of the *compound cystic* variety, the cysts containing a glairy, viscid fluid, or the cysts may present papillomatous ingrowths, or similar projections on their outer surfaces. In these two varieties nothing apparently is left of the ovary. The Fallopian tube may not be greatly altered.

Large *single cysts*, over which the Fallopian tube is stretched, develop from the *parovarium*—a foetal structure lying between the ovary and Fallopian tube. In this case the flattened ovary can usually be made out on the lower wall of the cyst.

*Dermoid cysts*, full of fatty material, and containing hair, teeth, and other representatives of organs, or parts of organs, may develop in the ovary. They are usually smaller than the cysts already described.

*Graafian Follicle*.—A Graafian follicle, after rupture in menstruation, appears as a brownish rounded cyst not larger than a pea, the colour being due to hæmorrhage into the cyst. Later the colour changes to yellow—corpus luteum—from organisation of the clot and fatty degeneration of the epithelium. Later still the fibrous tissue formed contracts, and a scar results. The corpus luteum of pregnancy is larger, and may remain of some size until the end of pregnancy, whereas that of menstruation remains for a few months only. Small cysts of the ovary may result from simple “dropsy” or distension of a Graafian follicle.

To examine the *Uterus* further, it is advisable to remove it from the body along with a portion of the vagina. The method of procedure is practically the same as followed in removal of the male pelvic organs, cutting through the vagina low down, on the same level as the rectum and urethra.

Open up and examine the rectum as in the male.

If normal, dissect it away from the vagina. Then, if the bladder be still attached, slit up the *vagina* in the middle line posteriorly.

Note presence of lacerations or ulcerations of its surface. If discharges be present note whether they come from vagina or os uteri. Note amount of projection of the *cervix uteri*, which is proportionately longer than normal in “conical cervix” and in infants.

*External Os*.—The nulliparous os is round and smooth; the parous os is transverse and fissured. The mucous membrane surrounding may be red and rough—“erosion”—or protrudes as a soft red mass, which may be mistaken for malignant disease. Occlusion of the os is rare.

Extensive destruction of the cervix occurs usually from malignant disease. *Cancer* may be squamous-celled, starting from the vaginal portion of the cervix, or tubular, starting from the mucous membrane lining the cervix, or from the glands opening on its surface. The surface of the tumour is usually very irregular, ulcerated, or warty.

Non-malignant *polypi* of the cervix project through it; they are glandular and small in size. *Fibroids* occur rarely in the cervix, though pedunculated uterine fibroids may project through it.

Open up the cervix and uterus in the middle line, and extend the incision into the Fallopian tubes.

Note the consistence and thickness of the uterine walls.

The normal cervix is fusiform in shape and rugose on its inner surface. Distinction of cervix from uterus is lost in cases of recent delivery. It may be elongated or hypertrophied.

The body of the uterus may show rounded, encapsuled *fibromyomata*—firm bodies, on section fasciculated and glistening. They may reach a



great size, and project internally or externally. They may become pedunculated. The *uterine polypus* is usually a pedunculated submucous fibroid.

Malignant disease of the uterus usually spreads from the cervix, though sarcoma and tubular cancer may commence at the fundus.

In *puerperal fever* the inner wall is shreddy and covered with foul discharge. Ulcers with sloughy base may be present, or abscesses may be present in the uterine wall. The ovarian veins may contain purulent thrombi.

The remaining structures on the posterior abdominal wall must now be examined. The *aorta* may show atheroma or aneurysm. In *psoas abscess* the psoas muscle is replaced by a thick-walled abscess cavity, which must be followed up to its origin in the bodies of the vertebræ. Clear the bodies of the *vertebræ* and examine them for fracture, dislocation, or disease.

THORAX.—The skin incision and method of clearing the front of the thorax have already been described (p. 460).

Cut through the costal cartilages obliquely downwards and outwards, beginning at the second. The knife is held nearly horizontal, so that when one cartilage is cut through, the haft falls on the next below, and thus the mediastinal contents are protected from injury. If the cartilages are calcified it is better to saw the ribs or divide them with the cutting bone-forceps well beyond the costo-chondral junctions.

Lifting the lower end of the sternum, separate the diaphragm from the costal cartilages, and free the sternum, keeping the knife close to its posterior surface. With the bone-forceps cut through the first rib cartilage, and separate the clavicle at its articulation with the manubrium.

In children the *thymus* gland, lying on the upper surface of the pericardium, behind the upper part of the sternum, and extending up into the thoracic inlet, must be examined, and any morbid appearance noted. Then proceed to examine the *lungs* and *heart*.

The *Pleuræ* are usually opened into in removing the sternum. Note the presence in them of fluid, general or localised, and its nature; exudation of lymph; adhesions, recent or fibrous, and their position. The fluid, as in other serous cavities, may be clear and straw-coloured in hydrothorax; turbid, containing flakes of lymph, in pleurisy; blood-stained in malignant disease of pleura, *e.g.* sarcoma, or in injury to the chest wall or lung. It may be purulent or putrid and offensive in empyema.

Remove and measure the fluid. Normally the pleura contains from half to one ounce of clear straw-coloured fluid, and its surface is smooth and glistening, showing in the adult a varying amount of carbon pigmentation.

*Lymph*.—On all serous surfaces, so long as lymph can be removed, leaving a smooth and shining, though congested surface, the inflammation is recent. When organisation commences it causes more or less incorporation of the lymph layer with the surface. Fully organised adhesions are practically non-vascular.

Next open the *Pericardium*. Pinch up a fold of it and incise it from base to apex. From the middle of this incision cut downwards towards the right auricle so as to expose the whole heart.

The contents may be similar to those found in the pleural cavities.

On the epicardium localised, white fibrous thickenings—"milk-spots"—may be present. Tuberculosis and malignant disease of the pericardium are rare and practically always secondary.



*The Heart.*—Examine the *heart* in situ. Note size, dilatation of individual cavities, and their proportion to each other. In great dilatation of the right auricle the venous sinuses are also distended.

Remove the heart. Lift it by the ventricles well out of the pericardial sac, and cut across successively the inferior vena cava, left and right pulmonary veins, pulmonary artery, aorta, and superior vena cava.

Open the cavities in the order in which the blood flows through them.

*Right Auricle.*—With the curved probe-pointed bistoury join the venæ cavæ, and from the centre of this incision cut into the appendix. Turn out thrombus, and notice whether it is distinctly adherent in the appendix, indicating ante-mortem formation. Note whether the foramen ovale is patent.

Examine the *tricuspid opening*. This normally, in the adult, admits three fingers. Take the cone diameter if desired. Vegetations are rarely present.

Place the heart on its posterior surface, and make an incision into the right ventricle, half-an-inch from and parallel to the septum ventriculorum. Holding the heart up by the margin of the cut pulmonary artery, allow a stream of water to flow into the artery, to test the competence of the valve. If thrombus be present, remove it before using the water test. If the water be retained the valve is competent. Take the cone diameter if desired. Continue the incision in the ventricle upwards into the pulmonary artery, cutting the latter close to the septum. The incision, if correctly made, ought to pass between two cusps. Note thickening, or, rarely and chiefly in infants, vegetations on the ventricular surfaces of the cusps.

Examine the *Right Ventricle*. Note the consistence, hypertrophy, or atrophy of the muscle. Note whether the sub-epicardial fat is invading the muscle. Note pallor, or a patchy yellowness of the muscle, the latter indicating fatty degeneration. The ventricle may contain ante-mortem thrombus, adherent between the columnæ carneæ, and, passing upwards into the pulmonary artery, in this situation showing the imprints of the sinuses of Valsalva. The wall of the ventricle varies in thickness normally from one-eighth of an inch at the apex, to one-quarter of an inch near the auriculo-ventricular groove.

*Left Auricle.*—Join the upper right and lower left pulmonary vein, and continue the incision into the auricular appendix.

Note dilatation, presence of ante- or post-mortem thrombus, thickening of the muscle, or endocardium. The endocardium normally is thicker than that in the right auricle. Press the finger down into the *mitral* opening, and note presence of stenosis, dilatation, thickening of the cusps, calcareous deposit, recent vegetations. The orifice, normally in the adult, admits two fingers.

Make an incision anteriorly into the *Left Ventricle* parallel to the septum ventriculorum, as in the case of the right ventricle, and in a similar way test the competence of the aortic valve. Take the cone diameters if desired.

Dissect away the *aorta* from the pulmonary artery, and extend the incision in the ventricle upwards into the aorta, passing between two of the cusps.

Examine the aortic cusps for vegetations, thickening, contraction, dilatation, adhesions in the angles, calcification; infective redundant granulations, ulcerating in their centre, destroying the cusps, and extending on to the wall of the aorta or ventricle. Slit up the *coronary arteries*, and note presence of atheroma or calcification in their walls and that of the aorta.

Note thickening and contraction of chordæ tendineæ, accompanying a similar change in the mitral segments, indicating old endocarditis.



Examine the wall for cloudy swelling, fatty degeneration and infiltration, myocarditis, infarct, and aneurysm.

Cloudy swelling causes pallor (often patchy) of the muscle, chiefly near the endocardium. Fatty degeneration and infiltration show appearances similar to those described in the right ventricle. If sections be made across the ventricle, near the apex, small white patches in the midst of the muscle indicate chronic fibroid myocarditis. Hæmorrhagic infarcts appear as brownish necrosed patches in the muscle. Aneurysms are localised dilatations near the apex of the ventricle, and result from myocarditis or infarct.

Note dilatation and hypertrophy of the walls. The ventricle in the adult measures about three inches from base to apex internally. The thickness varies from one-quarter of an inch at the apex to one-half to three-quarters at a short distance from the auriculo-ventricular groove. The papillary muscles and columnæ carneæ usually partake in the hypertrophy. Note presence of an abnormal persistent aperture in the septum ventriculorum. This is placed usually just below the right posterior aortic cusp.

If exact measurements of the orifices be desired the "cones" should be passed in the direction of the blood stream before slitting them up. The normal cone diameters are:—

Aorta, 0·9 in. to 1·0 in.	Pulmonary artery, 1 in. to 1·1 in.
Mitral, 1·1 in. to 1·2 in.	Tricuspid, 1·4 in. to 1·8 in.

Weigh the heart after examination, as now all blood-clot is removed. In the adult the weight varies from 9 ounces in the female to 11 ounces in the male. Externally it should measure at least 5 inches in length, 3½ inches in breadth, and 2½ inches in thickness.

*The Lungs.*—On opening the thorax the lungs, if absolutely healthy, would collapse. As a rule, however, they remain more or less voluminous. Collapse may be prevented by the presence of tough mucus in the bronchi, by vesicular emphysema, consolidation (interstitial or exudative), pleural adhesions, or other causes.

Passing the left hand into the left pleura, free any adhesions present, draw the lung forwards out of the thorax, and divide the root from behind. Remove the right lung in a similar fashion.

Weigh the lungs as soon as removed. The weight of the left lung varies from 15 ounces in the female to 21 ounces in the male, and that of the right lung from 17 ounces in the female to 24 ounces in the male.

Examine the surface. Note any deviation from the normal subdivision of the left lung into two, and the right into three lobes. In *emphysema* the parts affected are distended, pale, spongy, and contain little pigment. The condition is practically always present along the anterior borders. In excessive emphysema there may be large bullæ attached to the lung. Areas of *collapse* are dark in colour, non-vesicular, and depressed below the general surface. Firm areas raised above the general surface occur in lobular pneumonia, particularly tubercular, in infarction, and in stonemasons' lung.

In the very young, and in dwellers in the country, the lungs are pinkish in colour; but in all dwellers in towns, and in workers in mines especially, the lungs show a varying degree of black carbon pigmentation. This maps out the lobules on the outer surface, and follows the interlobular septa, vessels and bronchi on the cut surface. If excessive the condition is called anthracosis, or coal-miners' lung. The walls of the bronchi and vessels are never pigmented.



There may be pits or grooves on the surface, cicatricial in origin. At the bottom of the pit the lung substance may contain a calcareous, whitish nodule, surrounded by fibrous tissue, all that remains of an old tuberculous condition. Cicatricial grooves may also result from contraction of areas of collapse, or are due to new formation of fibrous tissue along the septa and its contraction. This may occur in syphilis and other conditions causing interstitial pneumonia.

Subpleural hæmorrhages occur in cases of asphyxia, mechanical or occurring in the course of other diseases, and also in toxic and chemical poisonings.

Slit up the larger *bronchi* for a short distance. In acute bronchitis they are brightly congested, and contain thick tenacious mucus.

Place the left lung on its posterior surface, with the *base* proximal and the root pointing to the operator's left; then with the long "section" knife make one sweeping cut from the lateral surface towards the root, so that the two halves open out on the root as on a hinge. Incise the right lung in a similar fashion, placing it with the *apex* proximal.

Note the colour and consistence of the lung, and the character of the fluid which escapes from the cut surface on pressure.

Normally the colour of the cut surface depends upon the amount of carbon pigment present; it crepitates between the fingers, and a small quantity of blood, frothy through admixture with air, can be squeezed out.

The surface may be pale in anæmic conditions. It may be deeply congested; bright red in acute, dull red in chronic or hypostatic congestions. If, in addition, the lung be friable, pneumonia is present or commencing. In chronic venous congestion the colour is a dull rusty brown, often patchy in distribution, and the surface dry, unless œdema accompanies.

The lung is tougher than normal, so that the fingers cannot readily meet through it, in collapse and in the chronic interstitial pneumonias.

The consistence of the lung may be increased by deposit in its spaces, or by permanent thickening of its substance.

The former condition is present in the *consolidation* of *pneumonia*, and may affect a whole lobe, as in lobar pneumonia, or scattered lobules or groups of lobules, as in lobular pneumonia. A lobule is the subdivision in connection with a terminal bronchus, and it may be recognised in the adult on the pleural surface as a polygonal area outlined by the pigment lying in the lymphatics in the interlobular septa. When consolidated these areas are devoid of air, the vesicles being filled with exudation or with catarrhal cells. In the early stages of pneumonic consolidation the part affected is congested, in the later stages it is paler in colour. In all stages the friability is greatly increased.

In *œdema* of the lung a large quantity of a watery, frothy, blood-stained fluid can be squeezed out on the cut surface. This does not stain the fingers to any extent.

In *capillary bronchitis* and simple broncho-pneumonia (lobular), muco-purulent fluid may be squeezed out of the smaller bronchi.

Demonstrate the condition of the larger bronchi by slitting them up. In *chronic bronchitis* their walls are thickened and they stand up prominently above the cut surface if the lung be not consolidated. In suspected thrombosis or embolism slit up the pulmonary artery, and follow out its main branches. In the former the blood-clot is adherent; in the latter, if recent, it is not adherent, and may be firm and pale in colour.

Lastly, examine the root glands as to carbon pigmentation, tubercular deposit, caseation and softening, or adenitis.

Some of the more common morbid conditions found in the lungs may now be referred to in greater detail.

In *lobar* or *croupous pneumonia* a large area, usually a lobe or more, is consolidated. There is recent pleurisy on the surface. The lung is very voluminous; o



section it is airless and dry. In *red* hepatisation it is of a dusky red colour, and the cut surface is indistinctly granular. Friability is extreme. In *gray* hepatisation—the next stage—the colour is a pinkish gray, the cut surface still dry and distinctly granular, the granules being the loosened plugs expressed from the vesicles by the retraction of their walls.

In *suppurating* pneumonia thick, creamy, sticky pus can be scraped from the cut surface.

In *simple broncho-pneumonia* (syn.: lobular or catarrhal pneumonia), as seen typically following measles in the child, the cut surface is flat and mottled. There are minute gray or yellow areas, sometimes branching, corresponding to the bronchioles and their immediately adjacent vesicles, which are filled with catarrhal cells and leucocytes. Between these the lung is congested, and in some cases—*fibrino-catarrhal* pneumonia—is consolidated from exudation of fibrin into the vesicles.

In the adult, *septic broncho-pneumonias* and *hypostatic pneumonia*, which often has a septic character, show larger consolidated areas raised above the surrounding congested and still vesicular or œdematous lung tissue.

The appearance of *tubercular broncho-pneumonia* is readily distinguished. The consolidated area is usually about the size of a pea, and raised above the surface. Several of these may coalesce to form larger areas. They are yellowish, firm, and may show softening in their centres. In the child the whole lung is found studded with these nodules, and no excavation may exist. In the adult, excavation is present at the apex, the broncho-pneumonic tubercles coalescing to form consolidated areas which break down in their centres. All stages of the process may be followed in the same lung.

Old cavities are distinguished by their smooth fibrous walls; recent cavities by their rough irregular walls, formed by caseating tissue.

*Acute Miliary Tuberculosis*.—*Miliary gray tubercles* may be scattered uniformly throughout the lung, in which case the bacilli have been disseminated by the blood-vessels; or they may be clustered along the septa, adventitia of vessels, or bronchi, following the lines of the lymphatics.

In the former there is also generalised miliary tuberculosis throughout the body; in the latter this does not usually exist, and the spread is more a local one.

*Hæmorrhagic infarctions* occur as consolidated areas, full of blood, pyramidal in shape, with the base to the pleura, over which there may be localised pleurisy. If a section be made the occluded vessel of supply can usually be seen near the apex of the infarcted area.

If necessary, the upper air and food passages may now be removed for examination. Extend the median skin incision upwards nearly to the symphysis menti, dissect away the skin on either side, and remove the soft structures left in the superior mediastinum and neck, as far up as the level of the hyoid bone. Then pass a narrow-bladed knife upwards through the floor of the mouth and free the soft parts from the lower jaw-bone all round; or, if it be advisable, for the sake of appearances, to retain the anterior part of the tongue *in situ*, cut it across about its middle. Draw down the loosened parts through the opening made, then cut across the soft palate at its junction with the hard palate, and detach it with the pillars of the fauces from the base of the skull. Continuing to drag gently upon these, free the remaining attachments of the pharynx to the vertebral bodies, and remove all the parts in one mass.

Laying them down on a flat surface, with the posterior surface upwards, slit up the œsophagus and pharynx with scissors; then, in a similar manner, open up the larynx and trachea. Note congestion of the surfaces, ulcerations, diphtheritic exudations, and new-growths.

In the later period of life the cartilages of the larynx and trachea are frequently ossified, sometimes show calcification or necrosis, the last condition being usually of tubercular or syphilitic origin.

Next, turn the parts so that their anterior surface is upwards, examine the thyroid gland chiefly as to atrophy in cretinism or myxœdema, or enlargement in goître, or new-growth.



The cervical or mediastinal glands may either be examined at this stage or before removal of the cervical structures from the body.

The walls of the thorax and the spinal column are now exposed, and may be examined more particularly.

First, examine the portion of aorta remaining for atheroma, aneurysm, or contraction.

Removing this, note whether fracture or dislocation of the vertebræ exist, and, passing outwards, examine the ribs for fractures or exostoses.

In rickets the costo-chondral junctions are greatly enlarged, forming beaded projections—"rickety rosary"—which are most apparent on the inner surface.

*Sewing up the Cavities.*—The examination of the abdomen and thorax being now complete, it remains to restore the body, so far as is possible, to its former appearance.

Remove all fluids from the cavities, replace such organs as are not required for further examination, and pack the thoracic inlet with absorbent wool. Replace the sternum, and with a few stitches secure it in position; then carefully sew up the skin incision, so that no fluid can escape. This is best accomplished by using a continuous "out-and-in" suture, passing the needle through the skin always from within, and pulling steadily on the cord so as to keep the stitches tense. The closer the stitches the better the result. They should not be farther apart than half an inch. Tie the last stitch securely so that it cannot possibly slip.

Then remove all traces of blood from the skin surface, and place a layer of absorbent wool along the line of the skin incision, fixing it, if necessary, with a few turns of bandage. This will prevent soiling of the shroud.

*Head and Spinal Cord.*—Examination of the brain and spinal cord may proceed after the thorax and abdomen have been closed.

Where the *spinal cord* has to be examined, remove it before the brain. This order is of special importance when the course of ascending or descending degenerations has to be investigated.

Turn the body on its face, and, standing on its left side, draw the integuments as far as possible to that side; then, with firm pressure on the knife, and inclining it outwards, cut down on the left side of the vertebral spines, so as to separate with one sweep all the soft structures down to the laminae from occiput to sacrum. Passing to the other side, and drawing the integuments to that side, through the same skin incision and in the same manner clear the laminae of the right side from sacrum to occiput. Then saw completely through the laminae, near the articular processes. If this be properly accomplished the subsequent procedure is easy.

In the lumbar region cut through one of the interspinous ligaments and the corresponding ligamenta subflava; then inserting the closed points of the cutting bone-forceps between the spines, lever up the upper spinous process. The laminae generally separate easily, and then, grasping the loosened vertebral arch with lion-forceps, or with the left hand protected by a towel, pull upon it, at the same time snipping with the bone-forceps through the laminae which are not yet divided. The arch of the atlas vertebra is usually very hard and resistant.

Note any fracture of the vertebral arch or hæmorrhage into the surrounding tissues or into the spinal canal. In the dorsal region a layer of protective fat is usually found lying upon the dura.

Grasp the dura at the lower end of the cord with dissecting forceps, and



remembering that the cord does not extend beyond the second lumbar vertebra, cut it across well below that point; then free the dura from its attachments right up the canal, and cut across the spinal nerve-roots as far out as possible. While doing this, and lifting the cord from its bed, traction must always be kept up on the dura sufficient to keep the cord straight, any bending or crushing of it being avoided, as this might produce changes simulating disease.

Cut through the dura at its attachments to the margins of the foramen magnum, and, dividing the cord transversely, close to the skull, remove it.

Examine the cut surface. It should be clean cut, not bulging, and the gray matter distinct from the white.

Lay it on a flat surface, and slit up the dura in the middle line before and behind. Note congestion of membranes, thickening of dura, adhesion to the arachnoid, subdural hæmorrhage, tumour-growth, thickening or calcareous deposit in the arachnoid, presence of purulent lymph or tubercular deposits (miliary or caseating).

Tubercular meningitis occurs most commonly in the cervical and lumbar regions; in the former associated with and in most cases extending from a similar condition at the base of the brain. It appears as a fine granularity or mottling of the surface, best seen on the anterior surface. The anterior surface may be distinguished by the large longitudinal vessels running along the middle line, or by the shape of the anterior horn, and the distinct anterior fissure seen on the cut surface.

If the cord is to be examined microscopically, cut it across at frequent intervals, between the origins of the spinal nerves, and place it in 10 per cent. formalin solution.

*Brain.*—Incise the scalp from mastoid to mastoid across the vertex, first separating the hair along the line of incision. Detach the scalp and occipito-frontalis as far forward as the superciliary ridges, and as far backwards as the external occipital protuberance.

Note presence of hæmorrhage, bruising, or suppuration, external to or under the occipito-frontalis, and any injury or disease of the bone.

Carry the knife horizontally round the skull in a line passing just above the superciliary ridges, and passing through the occipital protuberance. Part of the strong fascia over the temporal muscles should be left above this incision.—The reason for this will appear later, when we come to close the head.

Then make a saw-cut through the skull in the same line, taking care not to let the saw pass through its whole thickness. The left hand or the hand of an assistant steadying the skull must be protected by a towel against injury from slipping of the saw.

“To complete the separation of the skull-cap use the mallet and steel chisel, breaking through the remainder of the inner table, unless a fracture of the bones of the skull is suspected, in which case it is better to use the saw more freely, even at the risk of injuring the membranes or the brain” (Woodhead).

Then lever up the calvarium and remove it, leaving the dura intact supporting the brain. This will not always be possible, for example in old people, in chronic alcoholics, or where the dura has become fixed to the skull from old inflammation. In children, also, the dura is usually firmly attached to the bone. In such cases remove it with the calvarium, cutting it through in the line of the saw-cut.

After removing the calvarium examine its inner surface for fracture, erosions, or prominences.

The anterior fontanelle should be closed at the twentieth month. The



cranium may be *thinned* in rickets, congenital syphilis, or hydrocephalus. Note the presence of Wormian bones in the lines of the sutures. It may be *thickened* in old age and the various forms of osteitis. Examine the surface of the dura; slit up the longitudinal sinus, and note whether thrombosis, recent or old, simple or purulent, be present. Then incise the dura in the line of the saw-cut, except in the middle line before and behind, and draw the lateral flaps thus made upwards towards the vertex. Examine its inner surface for pachymeningitis hæmorrhagica, or other condition, and then note the condition of the pia-arachnoid and cerebral convolutions. The arachnoid may be milky from old fibrous thickening, or in recent meningitis.

There may be clear, turbid, or purulent effusion in the pia-arachnoid. The convolutions may be flattened, and the sulci shallow where there is distension of the ventricles, abscess, or tumour of the brain. Note whether this is symmetrical. The convolutions may be narrowed and atrophied, and the sulci deep and wide, filled with fluid in old age, in chronic alcoholism, and in certain other diseases (*v. INSANITY*). There may be tubercles in the pia mater, extending chiefly in the neighbourhood of the Rolandic and Sylvian fissures. These must not be confounded with the Pacchionian bodies which are clustered at the vertex close to the margins of the longitudinal fissure.

In septic and other toxic conditions there may be petechial hæmorrhages in the pia-arachnoid—circumscribed, or more often, diffused.

The membranes may be lacerated in cases of compound fracture. In *hernia cerebri* a mushroom-like projection of brain substance exists, from which, frequently, pus can be expressed.

*Congestion.*—Passive engorgement having disappeared, as the heart has already been removed and the venous blood drained away, active congestion is noticed as distinct injection of the delicate vessels covering the convolutions.

*Removal of the Brain.*—Cut across the attachment of the falx cerebri to the crista galli of the ethmoid. Then insinuate the fingers of the left hand underneath the frontal lobes, and lift them up from the orbital plates. Detach the olfactory nerves from the cribriform plates with the handle of the curved probe-pointed bistoury, and then, still raising and supporting the brain, cut across the cranial nerves, the carotid arteries, and the infundibulum as far from the brain as possible. After dividing the fifth nerves, turn the brain to one side, so as to expose the tentorium cerebelli, which is to be cut through from before backwards, close to its margin. Repeat this on the other side. Divide the remaining cranial nerves and the vertebral arteries; then, supporting the brain with the left hand, or calvarium, gently lever up the spinal cord, which, if not already cut across, should be divided as low as possible, and remove the whole, separating it from the dura.

Weigh the brain. In the adult male the average weight is 3 lbs. 1½ oz.; in the female it is about one pound less.

Examine the *base of the skull* for hæmorrhage, intra- or extra-dural; abnormal prominences—osseous, cartilaginous, or fibrous; localised suppurations or adhesions, especially in the neighbourhood of the petrous bones, and note their relationship to internal or middle ear. Note any obvious morbid change in the cranial nerves. Slit up the remaining sinuses and note presence of thrombosis, simple or purulent, particularly in the lateral sinuses, where it may be caused by mastoid disease, and may be attended with cerebellar abscess.



In *acromegaly* or *myxœdema* examine carefully the pituitary body.

Remove the dura and examine the base for fractures.

If the orbits or nasal passages and adnexa are to be examined, the portions of the base covering these must be mapped out and removed with the chisel and mallet.

*Examination of the Brain.*—If accurate localisation of a lesion be desired, the vessels of the brain should be injected with strong formalin solution, and the brain laid in a similar solution for about a week to harden.

In most cases the brain may be examined at once.

In the first place, examine the *base*. Note variation in the arrangement of the basal vessels, and atheroma or calcification of their walls. Look for thickening of the membranes by fibrous tissue, or for the presence of lymph in the membranes veiling the structures at the base.

In *tubercular meningitis* the membranes contain greenish gelatinous lymph, as far forward as and covering the optic chiasma, and in removing the brain from the skull a large amount of turbid fluid escapes from the subarachnoid space. On opening up the Sylvian and longitudinal fissures, tubercles—little gray or yellow points—are seen along the vessels. These may also extend up over the Rolandic areas, and back over the surfaces of the cerebellum, pons, and medulla. They rarely extend up to the vertex. The lymph may block the foramen of Magendie, and the ventricles be dilated—the so-called “acute hydrocephalus.”

In *posterior basic meningitis*—which is usually in reality cerebro-spinal—a somewhat similar but yellower effusion is present at the base, extending from the infundibulum backwards over the anterior part of the under surface of the cerebellum, and over the pons, medulla, and spinal cord. The foramen of Magendie is practically always blocked in this condition, and necessarily the ventricles are dilated. “Chronic hydrocephalus” is caused by the blocking of this foramen by a fibrous membrane produced by such a meningitis. The ventricles may be so distended as to reduce the brain to a thin-walled cyst, and the third ventricle projects at the base as a translucent, rounded swelling, stretching the optic chiasma over it.

In general *suppurative arachnoiditis* the whole brain is covered with a layer of pus or purulent lymph, which may be both internal to and external to the arachnoid.

In all cases of lepto-meningitis the arachnoid becomes milky.

Note the presence of hæmorrhages on the surface, laceration or softening of the cortex, presence of scars, for example, over gummata, and localise them accurately.

Superficial *softening* most often occurs in the area supplied by the middle cerebral artery.

In cases of softening, whether internal or cortical, slit up the arteries concerned, and seek for thrombosis or embolism. These are most frequently found at the origin of the Sylvian artery or its first branch, especially on the left side.

Next examine the *interior* of the brain.

Lay it with the base upwards. Remove the cerebellum, pons, and medulla. Make several incisions into the cerebellum, then separate it from the pons by cutting across its peduncles. Note the condition of the fourth ventricle, and make transverse sections of the pons and medulla, leaving the sections connected in front by the membranes.

Turn the brain vertex upwards, and examine by horizontal slices, cutting from within outwards through the hemispheres, and leaving each slice attached to its neighbours by the membranes on the outer surface.

The centrum ovale may be soft and œdematous, or show numerous small points of congestion.

On reaching the level of the corpus callosum make a vertical sagittal incision for half an inch into the white matter about half an inch



from the middle line, and then cut outwards, exposing the lateral ventricles. This dissection may be sufficient to demonstrate the lesion, or subsequent transverse, coronal, sections may be made across the basal ganglia to determine more exactly the relations of a lesion to external or internal capsules or ganglia, and portions of brain may be removed to expose the horns of the lateral ventricles. The fourth ventricle has been examined in connection with the pons.

If the brain has to be preserved for further microscopic examination, it is better to make the incisions in the following manner—that of Déjerine. After removing the cerebellum and pons, separate the hemispheres, and laying each in turn on its outer surface, make one horizontal section just below the level of the corpus callosum, and then to expose the basal ganglia, make two vertical transverse incisions in the lower part of the hemisphere just behind the anterior extremity of the corpus callosum, and in front of its posterior extremity. These incisions expose most of the important structures in which lesions may occur.

In cases of recent hæmorrhage, after noting its extent and relations, the affected part may be placed under a gentle stream of water, and the brain substance washed away. This exposes the vessels, which should be examined for minute swellings—miliary aneurysms, rupture of which may have caused the hæmorrhage.

In old hæmorrhages there may remain a shrunken, brownish mass, or a cyst with pigmented walls containing a thin fluid.

Newly-formed masses in the brain may be of the nature of tumours—most commonly glioma or sarcoma—the glioma infiltrating and with ill-defined outline, the sarcoma circumscribed—tubercular caseous nodules, or gummata.

To examine the *middle ear* it is best to remove the whole temporal bone, and examine it by sections either at once or after decalcifying.

After examination of the brain is completed, return what is not required for further investigation, replace the calvarium and secure it in position by stitches through the divided temporal fascia. In an adult, short spikes may be driven into the diploë, in front and behind, attaching the calvarium rigidly to the base of the skull.

Then sew up tightly the flaps of the scalp so that no escape of blood can take place. It is well subsequently to place a layer of cotton wool along the line of the incision and secure all with a few turns of a bandage.

No trace of blood or discharge must be allowed to remain on the surface.

This concludes the post-mortem examination.

Occasionally the extremities have to be examined, for example, in cases of fracture, of aneurysms, of suppurations in the tissues round the bones or in the joints. In rickets, in congenital syphilis, and in other conditions affecting the development of bone, it is well to expose the head of the humerus, and, sawing it longitudinally, examine the epiphyseal line.

## PART II.

### A. PRESERVATION OF TISSUES

1. If retention of the natural colour of a tissue or organ be not desired, wash thoroughly in running water and suspend in 50 per cent alcohol or methylated spirit, distending cavities *loosely* with horse-hair or cotton wool. Change the spirit at the end of a week, and again after a



similar period, if it be decolorised. At the end of three or four weeks mount permanently in 75 per cent alcohol or methylated spirit.

Or, place in 5 per cent solution of commercial "formalin," or of its equivalent, a 40 per cent aqueous solution of formic aldehyde. Change if necessary, and mount in a similar solution or in methylated spirit.

A simple mounting fluid for organs which contain fat is—

Formalin, 5 parts.  
Glycerine, 30 parts.  
Water, 65 parts.

2. If the natural colour is to be preserved, use a method such as the following, adapted from Professor Jores. Prepare the tissue as it is to be finally mounted, wash away excess of blood, and place in the following solution :—

Formalin, 8 ounces (10 per cent).  
Sod. sulphate, 700 grains.  
Magn. sulphate, 700 grains.  
Sod. chloride, 350 grains.  
Water to  $\frac{1}{2}$  gallon.

Dissolve the salts in the water and add the formalin or its equivalent solution of formic aldehyde. Inject the solution into the vessels in large organs or tumours so that it may reach the central parts, and fill hollow organs.

Retain in this for forty-eight hours.

Place in strong methylated spirit for twenty-four hours, changing if necessary. The spirit restores the blood colour which had been altered in the formalin solution.

Preserve in pure glycerine 2 parts, water 3 parts, changing if necessary. To the final mounting fluid 1 per cent of formalin may be added to prevent growth of fungi, but this is not absolutely necessary.

These directions may be taken as applicable to most tissues and organs. In the case of hollow organs, such as intestine, a much shorter time is necessary, and the action of the spirit should be carefully watched, as it is apt to decolorise the specimen.

With a little practice and patience excellent results are attained.

#### B. PREPARATION OF TISSUES FOR THE MICROSCOPE

*Necessaries.*—Cathcart microtome and accessories; methylated ether; mucilage of gum, B.P.; picro-carmin (Ranvier's); Ehrlich's hæmatoxylin; Farrant's glycerine mounting fluid; eosine in water (0.5 per cent); absolute alcohol; clove oil; Canada balsam dissolved in benzole; two needles in handles. Clean slides and cover-glasses.

Other reagents or apparatus will be mentioned as they are required.

A tissue can be examined under the microscope when a thin layer of it or its elements, penetrable by reflected light, is obtained. Examination of the surface of an object, using direct illumination without the mirror, is seldom had recourse to in pathology.

A thin layer may be secured by scraping the cut surface, or teasing out a small portion of the tissue with needles, but the most common procedure is to cut thin sections, either by freezing or embedding methods, and subsequently to stain them by suitable reagents which accentuate their component parts. Numerous methods of attaining this end have been recommended, but only such as are in common use, and have been thoroughly tested, can be described here. For others the reader is referred to pathological text-books, such as those by Sims Woodhead, Mallory and Wright, or to the *Pathologist's Vade-mecum*, by Lee.



*Examination of Scrapings of Fresh Tissues.*—This method has now gone out of fashion, but as it gives good results, and as it is easily carried out, requiring no special apparatus, it merits notice.

Make a fresh cut into the tissue or organ, squeeze out excess of blood if necessary, and with the edge of a knife scrape off some of the fluid from its surface. Transfer a little of this "tissue-juice" to a clean slide and mix with a drop or two of fresh Ranvier's picro-carmin. Gently lower a clean cover-glass on to the mixture, and let it sink by its own weight so as to produce a thin transparent layer of the fluid. Allow the stain to act for a few minutes and then examine. Nuclei of cells are stained with the carmine, protoplasm in varying shades of brown and yellow.

If after five or ten minutes the nuclei are not clearly stained, remove excess of fluid from the slide with bibulous paper, and paint a thin layer of vaseline round the cover-glass to retard evaporation. Examine again in an hour. No mounting medium is required, as the preparation is not permanent.

No more recent method gives a better idea of the natural appearance of the component cells of tumours or other tissues.

*Examination of Sections of Tissues.*—To obtain the best and most permanent results the tissue must be "fixed" before sections are made, although sections sufficiently good for diagnostic purposes may be got by cutting thin sections of fresh tissues with the freezing microtome, floating these on to a slide, staining for five to fifteen minutes in picro-carmin or in logwood, and mounting in Farrant's glycerine medium.

The ordinary stages in the preparation of permanent sections are—  
1. Fixation; 2. Hardening, embedding, and section-cutting; 3. Staining and mounting of sections. Several alternative methods of carrying these out have to be described:—

1. *Fixation of Tissues.*—A "fixative" is a reagent which penetrates and kills a tissue quickly, preserves the tissue-elements, and particularly the nuclei, in the condition in which they are at the moment when the reagent acts upon them.

For most purposes one may make a choice of four reagents—(a) formaldehyde, (b) formaldehyde and alcohol, (c) alcohol, (d) corrosive sublimate.

(a) *Formaldehyde*, or formic aldehyde, is sold in a standard solution of 40 per cent strength in water. A proprietary preparation of this strength is on the market, under the name of "Formalin"; it costs more than the other solution, but its action is identical.

A further dilution of 5 to 10 per cent—i.e. 2 to 4 per cent of the gas—of this standard solution is used for fixation of tissues, and tissues so fixed may be cut forthwith with the freezing microtome or after embedding.

Small pieces of tissue up to one-sixth of an inch in thickness are sufficiently fixed in three to six hours. The process may be greatly accelerated by gently heating to blood temperature.

Formaldehyde also hardens tissues, making albuminous bodies firm, but not precipitating them.

All tissues save those in which micro-organisms are to be demonstrated may be fixed with this reagent.

(b) *Alcohol.*—Except for fat-containing tissues this is the most useful general fixative, and gives specially good results where logwood is used for staining, or where bacteria are to be demonstrated.

Pieces of tissue not thicker than one-sixth of an inch, and of an area not greater than three-quarters of an inch, are placed in large excess of 70 to 80 per cent alcohol for a few hours, and then transferred to 93 to 95 per cent alcohol for at least two days. The tissue should be supported in the upper part of the alcohol by means of cotton wool. It has the additional advantage of hardening the tissue.

Ordinary methylated spirit is of a strength of 93 to 95 per cent, and acts as well as pure alcohol.

(c) *Formaldehyde (Formic Aldehyde) and Alcohol.*—Ten per cent of the solution of formaldehyde or "formalin" is added to absolute alcohol or methylated spirit.




Pieces of tissue of the size already mentioned are fixed in this medium in a few hours, and at the same time are so hardened and dehydrated that they may be passed on very rapidly to paraffin or celloidin. The alcohol dissolves out any fat contained in the tissues.

(d) *Corrosive Sublimate*.—The solution is a saturated one (7·5 per cent) in normal salt solution (0·75 per cent sodium chloride).

This reagent is very generally applicable. Nuclei stain well, fat and bacteria are preserved and perfectly fixed, so that they stain well.

Pieces of tissues up to one-half of an inch in thickness, fix throughout in eighteen to twenty-four hours. After fixation they must be left in running water for twenty-four hours to remove excess of the fixative. They are then transferred to gum and sugar, *i.e.* a mixture of 4 parts gum and 3 parts simple syrup, or if the tissue is to be imbedded in paraffin or celloidin, the procedure is as follows:—

Pass the tissue through successive strengths of 30, 60, and 90 per cent alcohol or spirit. To these add sufficient tr. iodi. to give a distinct brown colour. The iodine combines with any mercuric chloride left, producing the iodide, which is more readily dissolved out than the perchloride. If perchloride be left in the tissues it appears in the sections as disfiguring black crystals. The tissues show less shrinkage if they are gradually dehydrated, being left in each strength of alcohol for at least twelve hours. From the pure spirit, or 90 per cent alcohol, they are transferred to absolute alcohol to complete dehydration, and thereafter the stages are the same for all methods.

After twelve hours transfer from absolute alcohol to an agent such as cedar oil or chloroform, which will mix with alcohol and dissolve paraffin. Leave in cedar oil until transparent, or in chloroform until the tissue sinks, then transfer to paraffin, melting at 46° C., or about 116° F.; change after two hours into paraffin, with a higher melting-point 51° C., or about 123° F., and leave until all odour of the cedar oil or chloroform has disappeared. To keep the paraffin melted a small water-bath, regulated at about 52° C., or 125° F., is required. The initial cost is not great. After penetration is complete embed in freshly-melted 51° C. paraffin in a paper box, or in the -shaped adjustable apparatus sold with the microtomes.

Cool rapidly, holding the whole under cold water as soon as the surface of the paraffin has solidified, so that the paraffin may not form large crystals which might injure delicate tissues.

*Embedding in Celloidin*.—From the absolute alcohol transfer to a mixture of equal parts of alcohol and ether over night, then transfer to a solution of celloidin, in equal parts of alcohol and ether, of the consistence of thin cream. Cork this up and leave for twelve to twenty-four hours. Then transfer to a solution of the consistence of thick syrup for other twelve to twenty-four hours, and mount on a block of wood or cork of suitable size. Place a layer of thick celloidin solution on the block, put the impregnated tissue on this, and paint on a layer of celloidin all round and over it. As this dries apply other layers until the tissue is well supported. As soon as the whole attains some firmness transfer to and keep in 80 per cent methylated spirit, which hardens the celloidin.

2. *Section-cutting*.—(a) From gum and sugar, or from 5 to 10 per cent formaldehyde. Use a Cathcart ether-freezing microtome.

Remove excess of fluid from the tissue, place in a drop of mucilage of acacia B.P., on the zinc plate, and apply mucilage all round to support it. Freeze until the whole is of the consistence of cheese, and cut thin sections.

Instructions for using the microtome are furnished with each instrument.

Transfer the sections from the knife with a camel's hair brush to a bowl of cold, boiled water or normal salt solution, and mount on a clean slide. The advantage of using boiled water is that, as all the air is boiled out of it, no troublesome air-bells form under the section as it floats in the water.

(b) *Tissues imbedded in paraffin*.

Blocks prepared in the manner described may be cut with the Cathcart microtome, being fixed on to a special carrier by heating it gently, applying the block, and cooling the whole in water, or they may be cut with the



Cambridge rocking microtome. In both cases directions are supplied with the instrument.

(c) Tissues embedded in celloidin.

These may be washed out in water, soaked with gum and sugar, and cut like tissues embedded in gum and sugar, care being taken to keep the knife moist with 80 per cent spirit. Transfer the sections as cut to weak spirit. In the case of celloidin better sections are obtained if the knife is carried obliquely through the block, hence a long, heavy knife is to be preferred to the razor ordinarily used in the Cathcart microtome.

If mounted on blocks, the blocks are fixed in a special carrier, supplied, if desired, with the Cathcart microtome, or special microtomes adapted for celloidin may be employed.

### 3. *Staining and mounting of Sections.*

Sections cut by any of these methods except the paraffin method may be stained immediately on the slide or in a watch-glass.

Paraffin sections are floated out on the surface of boiled water just under the melting-point of the paraffin, and mounted on clean slides which may be albuminised.

*To Albuminise Slides.*—Take the white of an egg, shake it up thoroughly with nine parts of distilled water, add about 1 per cent of salicylic acid to retard decomposition, and filter.

Paint on a thin layer on the slide, place against a support, with the albumin downwards, to dry; when dry keep in a covered receptacle.

Albuminising is not absolutely necessary if perfectly clean slides be used.

A slide is placed obliquely under the paraffin section floating in the water, and the edge of the latter drawn on to it by means of a needle. The slide is then gently raised out of the water, bearing the section with it. It is dried and kept at room temperature over night, or preferably in a water-bath, the temperature of which is regulated near the melting-point of the paraffin in order to fix the section to the slide.

Before staining, the paraffin must be dissolved out with benzole, turpentine, or xylol, the solvent washed out with strong spirit, and the spirit washed out with water.

Celloidin sections are usually stained without removal of the celloidin.

## STAINING OF SECTIONS

*Picro-carmin.*—Cover the section with a few drops of Ranvier's picro-carmin and allow to act for a half to one hour. Remove excess with blotting-paper—do not wash—and gently drop some Farrant's glycerine mounting medium on the centre of the section. Lower a cover-glass on to this, as already described in the preparation of a "Scraping of Tissues" (p. 482). The staining gradually improves.

*Hæmatoxylin.*—While picro-carmin gives very good results, a preference is shown by most workers for a method which will give a more rapid differentiation of tissues, and especially of nuclei. Such a method is that now to be described. It is a little more complex than the former, but with a little practice facility in its employment is readily attained.

As the active colouring agent in most hæmatoxylin stains is the same—hæmatein—it is only necessary for ordinary purposes to give the method of using one of the more dependable of the hæmatoxylin stains—that introduced by Ehrlich.

Ehrlich's hæmatoxylin may be bought ready for use, or may be prepared as follows:—Hæmatoxylin, 2 grains; absolute alcohol, 60 cubic centimetres. Dissolve, then add—glycerine, 60 cc.; distilled water, 60 cc.; glacial acetic acid, 3 cc.; ammonia alum to excess.

Expose to the light, frequently opening and shaking until the liquid acquires a deep port-wine colour. This usually requires two months. If carefully stoppered the staining powers remain constant for years.

Stain sections of "fixed" tissues for fifteen minutes.

Apply a 1 per cent solution of hydrochloric acid in water or spirit to limit the stain to nuclei. This changes the section to a distinct red colour. The hæmatoxylin at this stage is acid and not permanent, so that it must be rendered alkaline by washing in tap water for one to two hours, or for a few seconds in



water to which a drop of strong ammonia has been added. After using ammonia wash thoroughly in tap water.

The hæmatoxylin is now blue and practically permanent.

Counter-stain for one minute in 0.5 per cent solution of eosine in water, wash in water, pass through methylated spirit and absolute alcohol to dehydrate, clarify in clove oil, benzole, turpentine, or xylol, and mount in Canada balsam dissolved in benzole or xylol.

It must again be noted that sections stained in picro-carmin must be mounted in Farrant's medium only.

If picro-carmin sections be dehydrated in alcohol and mounted in balsam, the picric acid is dissolved out wholly. If, however, a trace of picric acid be added to the clarifying agent the colour will be restored, but the result is not very satisfactory except in the hands of skilled workers.

For ordinary purposes of diagnosis no other stains are required.

*Fat.*—To demonstrate the presence of fat, sections may be cut by any of the freezing methods detailed, and stained black with osmic acid or orange with sudan.

Osmic acid is used in  $\frac{1}{2}$  to  $\frac{1}{10}$  per cent solution in distilled water, a stock solution of a 1 per cent strength being kept in a black stoppered bottle.

Sudan iii. is employed as a saturated solution in 70 per cent alcohol. In both cases the stain is applied till the required colour is obtained. The osmic acid preparations are mounted in Farrant's medium, or de-hydrated and mounted in balsam; the sudan preparations can be mounted in Farrant's medium only.

For the special methods of staining applicable to nerve cells and fibres the reader is referred to Ford Robertson's *Pathology of Mental Diseases*.

### C. BACTERIOLOGICAL INVESTIGATIONS.

This subdivision of the paper should be read in conjunction with earlier articles on FLUIDS, BACTERIOLOGICAL EXAMINATION OF; MICRO-ORGANISMS; and MICROSCOPE.

Bacteria may be examined as they occur in tissues or fluids, or after separation from these by cultivation on suitable media, or by inoculation into animals.

This last expedient need not be referred to further than to impress upon the reader the fact that it may not legally be practised unless in a place specially registered for the purpose, and unless the investigator have obtained a special license under Act 39 and 40 Vict.

The routine method of examination may be summarised thus, taking the case of a pathological fluid:—

1. Examine films stained by "simple" and "differential" methods, using a high power (700 to 1000 diameters or more). In some cases this may suffice for diagnosis, but in most cases it is insufficient, and the individual organisms are "separated" out by cultivation methods, and then examined in pure cultures, (1) microscopically; (2) by their behaviour in different culture media; and (3), if required, by inoculation into animals. In this manner, sometimes easily, often only after a tedious investigation, the identity of bacteria is made out.

Only methods which require little time in performance, and a minimum of apparatus, shall be described in this article.

A. *Staining* for bacteria, and the deductions to be drawn from the results obtained.

Before staining can proceed thin sections of tissues or thin films of fluids must be obtained.

(a) The method of obtaining thin *sections* has been described in the last section of this paper, and here it is only necessary to repeat that tissues are best prepared for bacteriological staining methods by fixation in alcohol or in corrosive sublimate.

(b) The preparation of *films* has now to be described.

The cover-glass should be thin, preferably No. 1, for work with oil-immersion objectives (see article on MICROSCOPE), and circular, the latter being an important



point particularly for those comparatively unaccustomed to work with such fragile articles, as the square cover-glasses are more readily broken.

Boil these in 10 per cent solution of hydrate or carbonate of soda; wash thoroughly in tap water; drain and pass through strong mineral or acetic acid; again rinse thoroughly in water, and keep in a covered glass jar in a mixture of equal parts of alcohol and water containing a little pure ammonia.

Be careful not to touch cover-glasses at any stage with the fingers; when prepared in this way, they should be perfectly clean.

Pick up a clean cover-glass with dissecting forceps and wipe dry with a clean, *old* piece of linen or silk, then catch it at the edge with a Cornet's spring forceps.

1. In the case of discharges or fluids generally, make a small loop (one-sixteenth of an inch in diameter) at the end of a platinum needle, *sterilise* the needle by making it glow in a spirit or Bunsen flame; when cool, take up a loopful of the fluid and spread thinly and evenly over the whole surface of the cover-glass, working from the centre to the periphery.

If the discharge be thick, place the drop on one cover-glass and invert another upon it. Allow the fluid to spread out in a thin layer, or, if necessary, exert gentle pressure and *slide* the cover-glasses apart. Place each with the film side uppermost on a piece of white absorbent paper and allow to dry.

2. When making a film from a culture (see later), firstly, place a loopful of water, preferably distilled, on the cover-glass, pick out a minute quantity of the culture with a *straight* sterilised platinum needle, mix up with the water, and then spread out as before.

Drying may be hastened by waving in the air, or by holding the film high over a flame.

*Fixation of Film.*—Pass the cover-glass, film side up, through a spirit or Bunsen flame three times, at about the rate of the swing of the pendulum of an eight-day clock, pausing at the end of each swing.

The film is now ready for the *stain*.

The colouring agents used belong to the class of anilin dyes, and must be basic. They also act upon cells or tissues, if present, and in order to render the bacteria distinct, these less important objects have to be decolorised. When used in this manner the agent is called a "simple" stain, no other colouring agent being employed.

Under certain circumstances which will be explained later (*vide* Gram's stain, and Ziehl-Neelsen's stain for tubercle), certain organisms retain a stain more firmly than others, so that they resist decolorisation, and stand out very clearly. The decolorised organisms or tissues present may thereafter be stained with another reagent, so as to render the contrast still more distinct. Such a method of staining is termed "differential," and is of great use in determining the identity of organisms.

It will be evident from the foregoing that *over-staining with subsequent decolorisation* is the rule in bacteriology.

It is advisable to obtain the following stock solutions of reagents, with which all the ordinary investigations may be carried out. A few other re-agents which are not absolutely essential will be mentioned as occasion for their employment arises.

*Stains* :—

1. Saturated alcoholic solution of methylene blue.
2. Saturated alcoholic solution of gentian violet.
3. Saturated alcoholic solution of *basic* fuchsin.

*Vehicles or Mordants* :—

4. One per cent watery solution of caustic potash.

This is further diluted in compounding the stain.

5. Anilin water.

Place excess of pure anilin oil in a stoppered bottle; fill with distilled water, shake frequently, and filter twice before using, wetting the filter papers with water. The water takes up 4 per cent of the oil.

6. Five per cent watery solution of carbolic acid.

*Other re-agents* :—

7. Absolute alcohol.
8. Pure anilin oil.

This may be used for dehydrating films or sections instead of absolute alcohol, which is very apt to take up water from the atmosphere, particularly when used only at long intervals. The method of employment is described later.

9. Pure sulphuric acid.



This may be kept in 25 per cent strength.

10. Glacial acetic acid.

These reagents should be preserved in glass-stoppered bottles.

11. Xylol.

12. Canada balsam in xylol.

*Accessories :—*

13. Platinum needle in handle.

14. Cornet's forceps.

15. Glass slides.

16. Spirit lamp or Bunsen.

17. Small cubic-centimeter measure.

18. Filter paper.

19. Six watch glasses.

When required the reagents are prepared and used thus :—

*All compounded stains must be filtered immediately before use.*

1. Methylene blue. *Læffler's alkaline methylene blue.*

Stock solution of methylene blue, three parts.

Aqueous solution of caustic potash (1·10000) ten parts, mix and filter.

In the case of the caustic potash, add ninety-nine parts of distilled water to one of the stock solution to make the 1·10000 solution.

The made-up stain keeps for about three weeks.

*Method :—*

Stain a film for five minutes ; a section for ten minutes.

Wash in water.

Decolorise in acetic acid, three drops to half a pint of tap-water.

Wash again, preferably in distilled water.

Dry partially with filter paper.

In the case of a *film* complete dehydration by holding high over a flame, and mount in balsam forthwith.

In the case of a section.

Dehydrate rapidly in absolute alcohol.

Clarify in xylol.

Mount in balsam.

*Or*

Remove excess of water with filter paper.

Apply a drop or two of pure anilin oil until perfectly transparent.

Wash in xylol and mount as before.

Films may also be dehydrated by these methods.

*Risks.*—Absolute alcohol is apt to decolorise a methylene blue preparation too much, so that it is well not to remove too much of this stain with the acetic acid.

*Alternative Stain.*—Thionin blue, 1 gramme in 100 c.c. of a 2½ per cent solution of carbolic acid, may be substituted for alkaline methylene blue, and employed in the same manner. This stain is more permanent and resistant and is preferred to methylene blue by many. Before using, dilute with two parts distilled water.

2. Gentian violet :—

Stock solution of gentian violet, one part (11 per cent).

Anilin water, nine parts (89 per cent).

The made-up stain keeps for six to eight weeks.

This is a very useful stain, and may be used both as a "simple" and as a "differential" stain.

(1) Simple staining with gentian violet.

Stain as with methylene blue.

Decolorise with methylated spirit or alcohol until of a pale gray colour.

Dehydrate and mount in balsam.

(2) Differential staining with gentian violet.

*Gram's method :—*

Stain as before.

Wash off excess in tap-water.

Apply Gram's fixative solution :—

Iodine, 1 part (gramme).

Potass. iodide, 2 parts (grammes).

Water, 300 parts (cub. cent.)

For one half minute for films, two minutes for sections.

Decolorise in methylated spirit, or 70 per cent alcohol.



Contrast stain in 0.5 per cent watery eosine (this is not absolutely essential).

Dehydrate and mount in balsam.

It is supposed that the proteid of certain organisms forms with the gentian violet and the iodine a compound insoluble in alcohol.

A modification of this method which sometimes gives better results, particularly in the case of sections, may be described, as it can be carried out with the agents at our disposal. It may be used in all cases in which Gram's method is indicated.

*Weigert's method* :—

Stain with gentian violet.

Wash in 1:600 solution of common salt.

Dry with filter paper.

Apply the iodine fixative solution.

Dry with filter paper.

Decolorise, and at the same time complete dehydration with anilin oil, three parts, and xylol one part.

Wash in xylol and mount.

### 3. Fuchsin. *Ziehl-Neelsen's method*.

This stain is used especially for demonstration of the tubercle bacillus.

Stock solution of fuchsin, 1 part.

Five per cent carbolic acid, 8 parts.

Or add the fuchsin solution to the carbolic acid in a test tube until precipitation begins. On heating, the turbidity disappears.

Filter some of the stain into a watch-glass.

Heat gently until all turbidity or surface metallic shimmer disappears, then drop in the film or section, face *down*.

Continue heating gently, until a bubble of steam rises, then keep hot for five minutes.

Remove the film or section.

Wash in tap-water and decolorise thoroughly in spirit or alcohol.

Complete decolorisation in 25 per cent sulphuric acid, allowing to act for five minutes.

Wash in tap-water.

For contrast stain use *alkaline methylene blue*, or aqueous methylene blue, made by adding a few drops of stock solution to distilled water until a deep blue is obtained.

Dehydrate, clear, and mount.

It is important to commence decolorisation with alcohol, as this affords a fairly accurate means of differentiating the tubercle bacillus from other "acid-fast" (acid-resisting) organisms.

*Spores*.—If the above stain be allowed to act longer—fifteen to twenty minutes—it may be used for demonstrating spores in organisms which propagate by that method, weaker sulphuric acid (1.5 per cent) being used to decolorise.

*Flagella*.—For methods of staining flagella the reader is referred to any of the text-books on practical bacteriology.

There is a considerable number of diseases in which one may hope for help in diagnosis by adopting the above procedures—for example, suppurations, abscesses localised or spreading, gonorrhœa, periostitis and osteomyelitis, erysipelas, pneumonia, tuberculosis particularly pulmonary, leprosy, influenza, anthrax or malignant pustule, acute spreading gangrene, tetanus, plague, actinomycosis.

Although given a list of such favourable length, the reader must not conclude that, provided the staining methods be carried out correctly, he will be able therefrom to formulate a diagnosis in every case he examines, conditions clinically similar being frequently caused by bacteria which are quite dissimilar. Still, in the majority of cases one may expect assistance, and in very many one can make an accurate diagnosis from such examinations.

In abscesses one may expect to find most commonly cocci singly, in clusters, or in chains. The films should be stained with a simple stain, or by Gram's or Weigert's methods. The pyogenic cocci retain the stain by the latter methods. To determine their identity they must be separated by cultivation on suitable media. Chains (streptococci) are usually found in acute, rapidly-spreading abscesses.

Suppurations occurring during the course of pneumonia and typhoid, whether of soft parts or bone, may be caused by the organisms associated with these diseases. The diplococcus of pneumonia (Sternberg and Fränkel) may often be readily distinguished by staining methods, its double lanceolate shape and capsule marking it out from other cocci present, especially if stained by Gram's method; but



if bacilli are found in abscesses during typhoid fever one must not conclude without very careful investigation, for which great experience and special methods are required, that these are the bacilli of typhoid, as many other organisms may be present. In empyema the diplococcus of pneumonia should be looked for (see vol. viii. p. 505).

In periostitis and osteomyelitis bacteriological investigation of the pus is usually not carried out to diagnose its presence, as that depends more on clinical data, but to determine its character so as to direct treatment. The most common organism found is the staphylococcus pyogenes aureus, but streptococci may be found, and their presence in certain cases may call for the use of anti-streptococcic serum.

Where the suppuration is tuberculous in origin one may frequently by Ziehl-Neelsen's method demonstrate the presence of the tubercle bacillus.

*Gonorrhœa*.—When a suppurative urethritis with production of creamy greenish yellow pus occurs shortly after connection, one may hope by staining simply with methylene blue or thionin to find gonococci of characteristic double character in the protoplasm of the pus cells. This absolutely confirms the diagnosis, the only organism which might cause confusion—so far as is known—being a diplococcus occurring in puerperal cystitis, and this stains by Gram's method, whereas the gonococcus becomes decolorised. In later stages the bacteriological diagnosis cannot be so confident, and cultivation in Wertheim's medium has to be employed. This is composed of a mixture of human blood and agar. Its method of employment need not be referred to here.

In *erysipelas*, fluid from the vesicles at the spreading edge or obtained by puncture may be examined by staining or cultural methods, and found to contain streptococci.

*Pneumonia*.—The "rusty" sputum should be examined by "simple" stains or by Gram's method to demonstrate the diplococcus with its capsule.

*Pulmonary Tuberculosis* (clin. *phthisis pulmonalis*).—The sputum from a suspected case mixed with 1·20 carbolic acid if desired, should be placed in a flat glass dish, and the small yellow points picked out and crushed between cover-glasses, or, if these be not prominent, a drop of the yellow part of the sputum should be taken. The films are stained by Ziehl-Neelsen's method. The tubercle bacilli stand out as red rods upon a blue ground. Other organisms present are stained by the methylene blue contrast stain.

A scraping from a *leprous* nodule or sections of it may also be stained by this method. The bacilli are found in clusters in the "lepra" cells, and also free.

*Influenza*.—The bacillus of influenza is very minute and difficult to demonstrate. The best stain to use is Ziehl-Neelsen's, diluted with four times its bulk of distilled water, and filtered. This should be allowed to act for half an hour, and subsequent decolorisation in weak acetic acid should not be carried far. The bacilli often occur in clusters, the long axes pointing mostly in one direction, and they show end-staining, the central part of the rod being only faintly stained, so that they may resemble minute diplo-forms.

*Anthrax*.—The bacillus is of large size and thick, and may be demonstrated by Gram's method in sections of, or in fluid from, the vesicles on the raised edge of the malignant pustule. Cultivation methods should also be practised.

*Acute Spreading Gangrene*.—The most important variety of this is malignant œdema, and great care must be employed—as in the case of anthrax—to avoid inoculating one's self. Here again fluid from simple puncture, or obtained in a sterilised hypodermic needle, may be examined for the bacillus of malignant œdema, which may present an oval spore lying near the centre of the rod. This must in addition be examined by anaerobic culture methods.

In cases of *tetanus* the discharge from the primary wound may be examined by staining, but the bacillus is not always found in this way. Cultural methods should always be tried.

*Plague*.—Fluid obtained by hypodermic puncture is examined by simple staining methods, and by culture on agar. Here again excessive caution is demanded. Buboes, when punctured, usually break down early. In pneumonic plague the sputum may show both plague bacilli and Fränkel's diplococcus, and it may be difficult to distinguish between them, even though the former does not stain by Gram's method. The plague bacillus is usually somewhat oval, and shows marked end-staining.

*Actinomycosis*.—The pus is thin, and contains minute yellowish points, which should be picked out and examined after staining by Gram's method. Usually an irregular felted collection of filaments is found, in which dichotomous branching may be distinguished.



These are some of the diseases which may be met with by the practitioner, in which bacteriological investigation by staining methods assists diagnosis.

B. Investigation by *cultivation* on suitable media, such as beef-broth, agar-agar, peptone gelatine, glucose agar, and blood serum.

*Beef-broth*, which is also the basis of agar-agar and peptone gelatine, is made as follows:—

Place in an enamelled pan  $\frac{1}{2}$  lb. (500 grammes) of minced *lean* beef. All visible fat must be removed. Add rather more than one litre (35 oz.) of water, and macerate for an hour at a gentle heat considerably below boiling-point, then bring slowly to the boil. Allow to cool so that the fat solidifies, filter through several layers of muslin, and make up to one litre. To this beef-extract, which is simply a solution of the flesh extractives and salts, and is closely analogous to normal urine, is added nutrient material in the form of digested albumin (peptone) and common salt.

Very good beef-extract may be made by dissolving 3 grammes of Liebig's extract of meat in one litre of water.

To make beef-broth (*bouillon*) take: peptone, 10 grammes (1 per cent); common salt, 5 grammes ( $\frac{1}{2}$  per cent); beef-extract, 1 litre; or smaller proportionate quantities. Dissolve with the aid of heat. Test the reaction of the result with litmus paper. If acid, render faintly alkaline with a few drops of 10 per cent solution of caustic soda. Again boil thoroughly, and filter through moistened filter-paper into a perfectly clean glass flask or into test-tubes. Plug with unmedicated cotton wool, and sterilise for an hour on three successive days in a Koch's steriliser or in a potato-steamer.

The broth is then ready for use.

A solid medium is, however, for many purposes more convenient, and to obtain this, agar or gelatine (finest gold medal) is added to the beef-broth. The former is for incubation at high temperatures, the latter for cultivation at room temperatures. The agar and gelatine of themselves have no nutrient value.

The method of preparing a litre of each is described. If properly sterilised, they keep indefinitely.

*Agar-gelatine*, or shortly "Agar."

Agar-agar, 20 grammes (2 per cent).

Beef-broth, 1 litre.

The agar is cut up in small pieces and soaked for a quarter of an hour in about 1·500 acetic acid in water. It is then collected in a piece of muslin and washed thoroughly in tap-water until all trace of acid reaction disappears. This treatment facilitates solution.

Boil thoroughly in an enamelled pan, avoiding burning, until all the agar is dissolved. See that the reaction is still slightly alkaline; cool down to about 45° C., and add the white of an egg with the shell which have been beaten up with 100 c.c. of water. This water helps to make up for loss by evaporation. Shake up and boil again thoroughly, stirring the while to prevent burning, until the egg coagulates and separates. Filter *hot* through special agar filter-paper or through pure cotton-wool or "glass" wool. The former gives the better result, and use of a hot water jacketed filter renders the operation easier. Tubes may be filled and plugged at once, or the prepared agar kept in a plugged glass flask, the plug being covered with tin-foil.

Tubes and flask are sterilised as in the case of the broth. During cooling the tubes are inclined so that the agar solidifies with a large surface. Water of condensation collects in the bottom of the tube.

*Glucose agar* is made by adding 1 to 3 per cent of glucose to agar. The tubes are half-filled, and allowed to cool in the erect position.

*Peptone gelatine*, or simply "Gelatine."

Finest gold medal gelatine, 100 grammes.

Beef-broth, 1 litre.

Dissolve with *gentle* heat—preferably in a flask in the steamer, neutralise if necessary, clarify and filter. Fill tubes one-third full, and allow to solidify in the erect position.



The manufacture of the media, particularly of agar, presents certain difficulties, and if they be only occasionally required it is better to purchase them in tubes ready for use from scientific instrument dealers.

*Blood serum* can be very readily and easily prepared by any practitioner, and is useful for many purposes. The blood may be human or from the lower animals (cow or sheep).

Sufficient blood may be expressed from a placenta to make two or three tubes. With precautions as to cleanliness placental blood, or blood of the cow or sheep, is collected in a small beaker or flask, and after the clot contracts—say in ten hours—the serum is pipetted off into sterile tubes. These are plugged and supported in the steamer in an inclined position. The heat is applied very slowly, and not allowed to reach boiling-point, but only about 80° C., so as to coagulate the serum firmly. This requires one and a half to two hours. The tubes are then sterilised for half an hour on three successive days. The writer has found this method of preparation quite satisfactory, and the resultant solidified serum, though nearly opaque, gives quite as good cultural results as that prepared by other methods, and, moreover, no special apparatus is required.

*Loeffler's* blood serum, on which the diphtheria bacillus grows best, is prepared by adding two parts of separated blood serum to one part of beef-broth, to which 1 per cent glucose has been added. This is solidified as before.

For the preparation of other media the reader is referred to any textbook of bacteriology.

#### EMPLOYMENT OF THESE MEDIA

*Agar (or Blood Serum).*—Take three sloped tubes. Singe off the top of the wool plug, passing the lip of the tube through the flame. If the water of condensation be excessive remove the plug and pour it off.

To inoculate, take a loopful of the material with the sterilised and cooled platinum needle, and holding a tube horizontal, remove the plug by grasping it with the flexed, right, little finger, then passing in the platinum needle, avoiding contact with the walls of the tube, mix up the material with the fluid still remaining at the bottom of the tube. Then draw the loop gently back and forwards over the surface of the medium in a zigzag manner, so as to pass once over most of the surface. Replace the plug, and without recharging the needle repeat this process with the second and third tubes. In the second and third tubes the numbers of organisms ought to be so attenuated that separate colonies will develop on incubating at 37° C. for twenty-four hours.

For this, an "incubator," in which the temperature is kept at a uniform temperature of 35° to 37° C., or 98° to 99° F., by means of a suitable regulating apparatus, should be employed, and is essential for reliable work, though some cultures will develop at room temperature or by placing on a mantel-shelf above a fire. Portions of separate colonies thus obtained are to be picked out with the straight needle, and examined by "film" methods (p. 486).

*Gelatine.*—This medium may be used in the same way as agar, and incubated at room temperature, that is, from 18° to 22° C.

It is more often used to test colonies which have been separated on agar or serum. Pick out a little from the centre of a colony with the straight sterilised needle, stab this down through the centre of the solid gelatine, and incubate at room temperature.

Some organisms cause liquefaction of the gelatine by a peptonising action, *e.g.* staphylococci, anthrax, cholera; others grow in a characteristic manner, forming a heaped-up mass on the surface (Friedlander's diplobacillus), or sending out lateral processes into the medium (anthrax), or otherwise producing a characteristic appearance.

*Glucose Agar.*—The tube should be one-half full of *freshly prepared* glucose agar.

This medium may be used in attempting to separate anaerobic organisms,



other methods being too complicated for performance by any but skilled bacteriologists.

With a long, straight, sterilised platinum needle take a quantity of the discharge or fluid to be examined, and pass the needle deep into the medium. Replace the plug and cover with a tightly-fitting rubber cap. Repeat the process in two other tubes.

Incubate at 37° C. for forty-eight hours; then examine the growth which develops by the film method. If organisms containing spores be found, place the tubes in water kept at a temperature of 80° C. for half an hour. Then reinoculate tubes from each and incubate as before.

The exposure to a temperature of 80° C. should kill all non-sporing bacteria present.

The resultant cultures may contain any sporing, anaerobic organisms, such as tetanus or malignant œdema, which are often found together.

The reader must be warned, however, that these are very difficult to separate.

*Blood Serum.*—This is especially suitable for the diagnosis of *diphtheria*.

With the sterilised loop remove a little of the membrane from the throat in a suspected case, inoculate successively three serum tubes, and examine films after incubation for twelve to twenty hours. The diphtheria bacilli develop quickly on this medium, and if present, are readily recognised on staining with methylene blue or thionin blue. They also stain by Gram's method.

A dependable diagnosis cannot be made by simply examining a film made direct from the throat.

**Pregnancy.**

This subject will be described in the following sections:—

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**B. PATHOLOGICAL SECTION**

*See Vol. x.*

*See also* LABOUR, ABORTION, PUERPERIUM.

**A. PHYSIOLOGICAL SECTION**

**Changes in the Maternal Organs**

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As soon as an impregnated ovum becomes implanted in the uterine mucosa a series of remarkable changes sets in, not only in the generative organs, but in adjacent and distant structures, and indeed, the whole general economy becomes materially altered. These modifications may be conveniently divided into (1) local; (2) general.

### I. LOCAL CHANGES

The most important of these are necessarily in the uterus itself. At each menstrual epoch changes of hypertrophy and increased vascularity take place, but if impregnation does not occur, a destructive process sets in resulting in the menstrual discharge. If, on the other hand, conception occurs, the hypertrophic process continues, giving rise to among others remarkable changes in the size, capacity, and form of the uterus. The modifications which take place in the uterine mucosa, resulting in the formation of the deciduæ, have been described.

*Changes in the size, weight, capacity, and form of the uterus.*

The unimpregnated normal uterus measures 3 inches in length, 2 inches in breadth, and 1 inch in thickness. Some authorities state the measurements of length and breadth as rather less than these— $2\frac{3}{4}$  and  $1\frac{3}{4}$  inches. At full term of gestation the uterus has so grown that it measures from  $12\frac{3}{4}$  to  $15\frac{1}{4}$  inches in length, from  $9\frac{1}{2}$  to  $10\frac{3}{8}$  inches in breadth, and from 9 to  $9\frac{3}{4}$  inches antero-posteriorly. The circumference at the level of the Fallopian tubes is about 29 inches.

The weight of the virgin uterus is about  $1\frac{1}{4}$  ounces, that of the full term gravid uterus about 28 ounces.

The surface of the unimpregnated uterus measures  $5\frac{1}{2}$  square inches, and its capacity is 1 cubic inch. At the end of pregnancy the surface of the organ is 350 square inches, and its cubic capacity is 400 inches, so that the latter has increased over 500 times.

The *form* of the uterus also undergoes varying modifications as pregnancy advances. During the first three months the uterus retains its pyriform shape: the cervix is not much altered, and is still conical. Expansion occurs chiefly laterally, so that the uterus appears as a spherical body placed over a cervix almost normal in shape. Later, the uterus becomes expanded in its lower part, and by the fifth month it presents a more or less spherical form, the largest diameter being the vertical. At full term the uterus is described as ovoid or egg-shaped, the dome being at the fundus, the smaller end below. However, Spiegelberg has pointed out that the shape of the uterus during pregnancy is by no means uniform. Many deviations occur. The form depends on the primary shape of the organ, on the woman's position, on the position of the fœtus, and on the tension inside the uterus.

*Changes in the Position of the Uterus.*—Owing to increase in weight and size the uterus necessarily undergoes a change of position as gestation advances. It is very generally believed that during the first four weeks of pregnancy the uterus is situated somewhat lower in the pelvis, and some believe that the flattening of the hypogastrium is an important early sign of pregnancy. Tarnier, however, pointed out that this is not constant, and that very early the fundus rises above the level of the pelvic brim, while the cervix is situated at its normal level in the pelvis. At the end of the third month the fundus is a finger's breadth above the pubis; at the end of the fourth this distance has increased to rather more than two inches; at the end of the fifth to four inches; and at the end of the sixth



month it is on a level with, or a little above, the umbilicus. A month later the fundus is two inches above the umbilicus, and during the eighth and ninth months it continues to rise till its upper limit lies immediately below the ensiform cartilage. The greatest distance of the fundus from the pubis is attained about fourteen days before full time, for during the last two weeks there is commonly a marked descent, owing to the entrance of the lower pole of the uterus into the pelvis.

In multiparæ the abdominal wall is relaxed, so that the uterus projects farther forward, and the fundus does not rise so high as in primiparæ. In the latter, too, the descent of the lower pole occurs earlier. No doubt the rigidity of the uterus itself in primiparæ tends not only to preserve its upward growth, but furthers the early descent of the lower pole.

The situation of the impregnated uterus is normally somewhat oblique, being in the majority of cases slightly rotated, so that the anterior surface looks forwards and to the right and further, it does not occupy exactly the median position, deviating in most women slightly to the right side.

As to the causes of these deviations there is considerable difference of opinion, and many theories have been advanced to explain them. Some attribute them to the primary *set* of the uterus in the pelvis, some to the fact that women lie most commonly on their right side during sleep, some to the supposed comparative shortness of the right round ligament, some to irregular contractions of the uterine muscle, and others to distension of the rectum.

As gestation progresses the obliquities are maintained, and in the later stages are accentuated. The uterus grows upwards and forwards, so that in the erect position its long axis is in line with the axis of the brim.

In women who have had many children there occurs frequently a much more marked anterior projection of the uterus, and in some extreme cases the fundus is actually dependent.

At the end of pregnancy three-fourths of the anterior surface of the

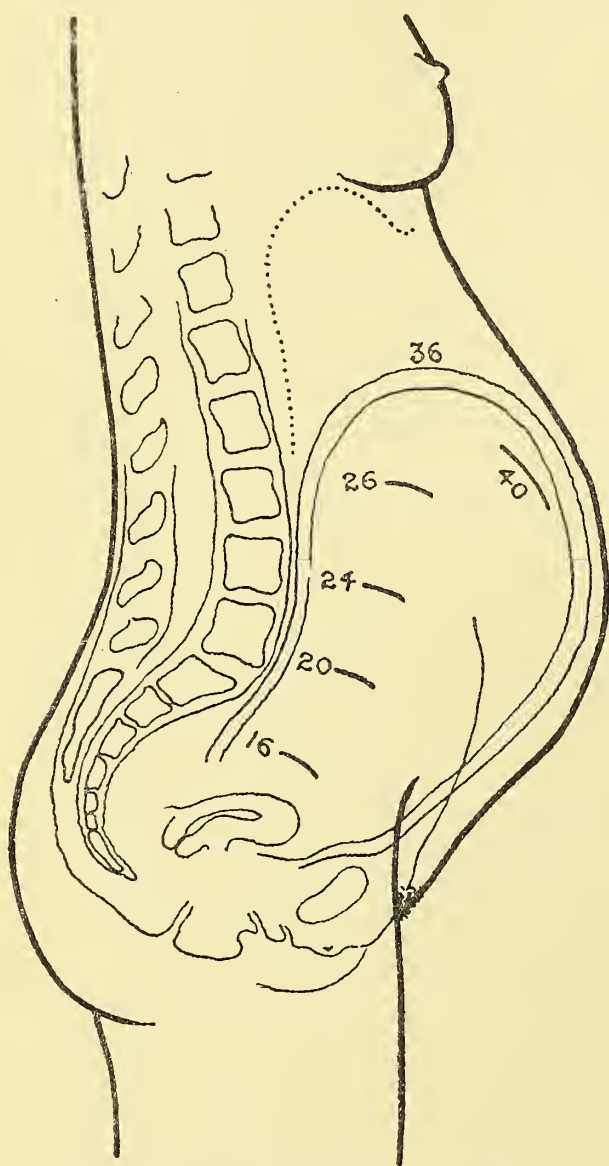


Diagram to illustrate the growth of the uterus during pregnancy.

uterus is in close relation with the abdominal wall, though sometimes omentum or small intestines may intervene. The lower fourth is in contact with the posterior wall of the bladder. The posterior surface of the uterus is in contact with the spinal column, and the coils of small intestine find room on either side. The fundus is in contact with the transverse colon and part of the greater curvature of the stomach, the anterior margin of the liver, and the xiphoid cartilage. The ascending colon is in relation



with the right border of the uterus, and the descending colon with the left.

*Changes in Structure.*—At one time it was supposed that the growth of the uterus was the result of mechanical pressure on the part of the enlarging ovum. If such were the case the uterine parietes would be much thinner at full term than in the non-gravid state. As a matter of fact, the thickness at full term is about the same as before pregnancy occurs, and the enormous increase in the size of the uterus is due to hypertrophy of the original muscular wall.

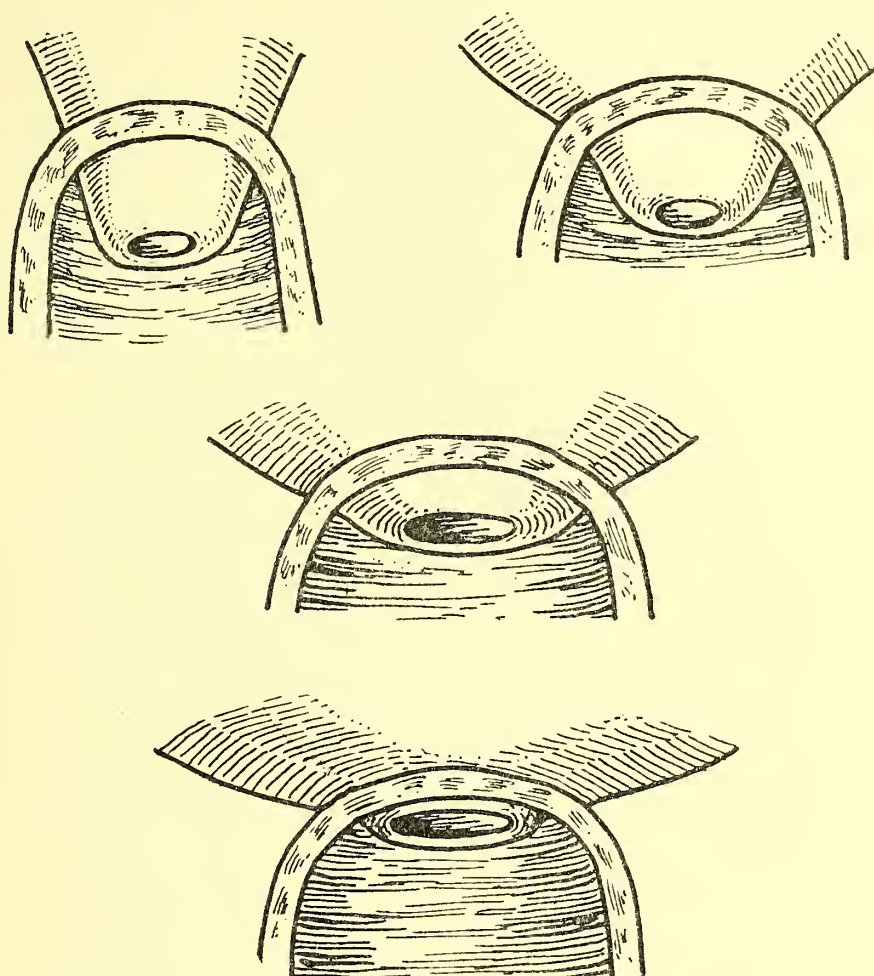


Diagram to illustrate the changes in the cervix during pregnancy.

There is, in addition, a distinct alteration in the muscular texture of the uterus, for one of the earliest signs of pregnancy is a softening and yielding of the wall, which strongly contrasts with the dense, unresistant pre-gravid character of the muscle.

The thickness of the wall varies in different women and at different parts of the same uterus, for example, at the placental site the muscle is thicker, and in the neighbourhood of the cervix it is thinner than in the virgin state. This hypertrophy is due to an enormous increase in size of the individual muscular fibres. They increase in length from seven to eleven times, and from two to five times in breadth. But besides the normally developed fibres which grow so greatly, there are in the uterine wall embryonic muscle-cells which, when pregnancy occurs, take on active development, and increase to a size commensurate with that of the pre-existing fibres.

At full term the uterus may be divided into three parts: above, the body; below, the cervix; and between these there is developed a portion of the muscular wall, which is much thinner and much less vascular than the body proper. This is



known as the lower uterine segment. It is about three or four inches in vertical extent, and is separated from the body by a distinct ridge known as "Bandl's ring," or the "retraction ring." During pregnancy this division has as its lower limit the internal os, but during labour, after the canalisation of the cervix has taken place, the lower uterine segment and the cervical canal are continuous. These parts of the uterus differ both in structure and in function. In the body the muscular fibres are arranged quite irregularly, in the lower segment chiefly vertically, and in the cervix chiefly in a circular manner. The body of the uterus is the only contractile part. The lower uterine segment and the cervix together are expansile.

During pregnancy the cervix uteri undergoes hypertrophic changes similar to those in other parts of the organ. At one time it was believed and taught that from the early months onwards there was a gradual shortening of the cervix, but it is now agreed that this diminution in length is virtual and not real. The apparent shortening is explained by the great softening of the cervical tissue which occurs, and to a certain extent to the altered direction and position of the cervix. There is, however, a real shortening of the cervical canal during the last fortnight of pregnancy, owing to the gradual dilatation of the canal from above downwards. Even in primiparæ in most cases the os externum is somewhat patulous during the later months, but in some it remains closed till labour sets in. In multiparæ the margins of the os are irregular, and it is always possible to introduce the tip of the finger even before the seventh month.

An examination of the muscular coat of the unimpregnated uterus reveals no definite arrangement of the muscular fibres. Helié has, however, shown that during pregnancy three distinct layers can be differentiated—an external, a middle, and an internal. The external layer, composed of alternating vertical and transverse fibres, covers the body and the fundus of the uterus, commencing at the junction of the cervix in front, and extending rather lower behind. At the fundus the lateral fibres diverge outwards on both sides, and are prolonged along the round ligaments, the Fallopian tubes, the ovaries, and the broad ligaments. The middle layer constitutes the bulk of the uterine wall. The fasciculi which compose it are of variable size, and cross each other in all directions. They surround the veins whose walls consist only of intima, and this arrangement affords an explanation of the arrest of hæmorrhage during the contraction after delivery. The internal layer is composed partly of circular rings of muscular fibres, which are especially well marked round the openings of the Fallopian tubes and round the os internum, and partly of longitudinal fibres arranged in a triangular fashion—one on the anterior and one on the posterior wall of the uterus. The base of the triangle is above and the apex at the os internum. Fibres of these three different layers pass from one stratum to the other, binding all into one continuous muscle.

The muscular coat of the cervix is composed chiefly of transversely circular fibres.

*Changes in the Peritoneum and in the Appendages.*—Owing to the gradual increase in size of the pregnant uterus the normal relations of the pelvic peritoneum are disturbed. The layers of the broad ligaments are separated; they share in the general hypertrophy, and are carried up by the growing uterus, so that instead of being horizontal they become vertical, or nearly so. As the result of this change of position the Fallopian tubes and ovaries are drawn towards the uterus, till at the end of pregnancy they lie alongside the uterus. The changes in the relations of the peritoneum in front and behind the uterus have not been definitely determined. Pollis's observations led him to believe that the normal level of the peritoneum in the non-pregnant condition was raised during pregnancy both in front and behind the uterus, whereas Webster, from an examination of frozen sections, found the inferior limit of the peritoneal pouches as low during pregnancy as before it.

The round ligaments increase enormously. They become four times their normal thickness, and they so increase in length that at the end of pregnancy they extend from near the umbilicus to the inguinal canal on each side. The utero-sacral ligaments also become enormously developed.



The ovaries are almost doubled in size, and assume nearly a vertical position. Ovulation is in most cases entirely suspended. The Fallopian tubes undergo the usual hypertrophic changes, increasing in thickness and in length. The epithelial lining becomes denuded of its vibratile cilia.

*Changes in the Arteries and Veins of the Uterus.*—The arteries of the uterus increase in size, in length, and in number. The change in their length is not due to their becoming less tortuous, for Jacquemier has found that they are more tortuous at the end of pregnancy than in the non-pregnant state. The ovarian arteries become about the sixth of an inch in diameter, and the uterine arteries are still larger. The branches on entering the uterus suddenly enlarge, and a very fine anastomosis is established between the two sides of the uterus. Those which pass to the endometrium subdivide freely, and end in a diffuse capillary network. The veins of the uterus become dilated into large sinuses which communicate with each other. The walls of these are intimately united with the surrounding connective tissue, and therefore when the venous channels are mutilated the walls do not collapse, but remain more or less gaping. These veins are devoid of valves, and in the middle muscular layer the wall consists of a single coat. The ovarian veins so hypertrophy that they are almost as large as the external or internal iliac.

*Changes in the Lymphatics and Nerves.*—The uterine lymphatic system undergoes hypertrophic changes during pregnancy. The whole intra-uterine expanse may be looked upon as one vast lymph sinus (Spiegelberg). The lymphatic channels, when they pass from the substance of the uterus, are collected into a plexus underneath the peritoneum, especially over the fundus and along the sides of the uterus. The lymphatics of the fundus and the body pass to the lumbar glands, while those of the cervix communicate with the pelvic glands. The exact changes in the nerves have not been accurately determined. The cervical ganglion increases in size till it is three or four times as large as in the non-pregnant condition. Some authorities, including Snow, Beck, and Hirschfeld, believed that the nerves remain of the same size as in the unimpregnated uterus. Robin held that there was an apparent increase of size, but that this was due to increase of neurilemma. Kilian believed the nerve tubes increased in length, but not in thickness; while Schroeder stated that they hypertrophied in every way, just as the lymphatics do.

*The Vagina and External Genitals.*—Changes of hypertrophy similar to those which occur in the uterus, though to a lesser degree, take place in the vagina and external genitals. The vaginal canal increases in length and in capacity. The submucous adipose tissue becomes less in amount, but there is marked development of the muscular coat and of the connective tissue. The blood-vessels increase in size and in number, and in consequence of the increased vascularity the mucous membrane assumes a bluish, almost purple, colour which by some is regarded as a very important sign of pregnancy. The mucous membrane of the vagina hypertrophies more than the muscular and external coats, with the result that the normal folds are exaggerated, so much so that frequently these protrude through the vulvar orifice as a violet-coloured swelling. The normal secretion of the vagina is increased, in some cases to such an extent that it forms an actual discharge. The cervix generally occupies a position somewhat to the left of the middle line, and it seems shortened owing to the hypertrophy of the mucous lining of the vagina and the ascent of the uterus into the abdomen. The external genitals become hypertrophied and softened, owing to the increase of vascularity and the development of the lymphatics. The



vulva, therefore, appears prominent, the labia being swollen, and the veins seem varicose, while the secretion from the sebaceous and sweat glands is much increased. In multiparæ the vulvar orifice gapes, while in primiparæ it appears smaller and retracted.

*The Pelvic Joints.*—To a certain extent the articulations of the pelvis share in the changes due to pregnancy. Zaglass held that there was a certain, though very slight, amount of movement in the sacro-iliac joints in the non-pregnant woman. During pregnancy, and especially during the last weeks and during labour, there is a marked serous infiltration of the joints and elongation of the ligaments, and in many women distinct movement of the sacro-iliac and pubic joints can be made out during labour. The sacro-coccygeal joint also becomes more movable.

*Adjacent Organs.*—The other organs in the pelvis undergo changes during pregnancy, partly due to excessive congestion similar to that which occurs in the uterus, and partly due to mechanical pressure. During the early months the pregnant woman suffers from irritability of the bladder, frequent micturition, and sometimes incontinence. These symptoms are due partly to pressure by the growing uterus, and partly to hyperæmia of the vesical mucous membrane. Towards the end of pregnancy these symptoms recur, owing to the sinking of the uterus; and in some cases retention of urine results from the pressure of the presenting part on the urethra.

Nearly all pregnant women suffer from constipation. This, if not due to persistence of habit, is the result of pressure of the gravid uterus upon the sigmoid flexure, interference with the normal peristalsis, and the limited action of the abdominal muscles.

Œdema of the lower extremities and a varicose condition of the veins are very common during pregnancy. These, during the later months, are the result of increased intra-abdominal pressure, while in the early months the same changes may occur merely from the slowing of the blood current consequent on the dilatation of the pelvic veins. Pains in the lower extremities are referred to pressure upon the nerve trunks from the lumbar and sacral plexuses.

*The Abdominal Wall.*—The distension of the abdominal walls during pregnancy leads to the production of the “*striæ gravidarum*.” These are bluish red lines which become most conspicuous in the later months, and are found over the lower part of the abdomen, and especially the sides. After labour they persist, and gradually become white in colour. These striæ are the result of the stretching of the deep layer of connective tissue of the cutis. During pregnancy these lines are also seen on the nates, the thighs, and the breasts. They are not peculiar to pregnancy, for similar marks may appear on the abdominal wall when it is rapidly stretched, as from a tumour or ascites. Other pigmentary changes in the skin occur, but they are not constant. The most common is the appearance of what is called by some the “*linea nigra*,” a dark-brown line which runs from the symphysis pubis to the umbilicus, or even beyond it. Patches of discoloured skin sometimes appear on the face, especially on the forehead; but these pigmentary changes in the skin are of no diagnostic value in pregnancy.

By the fifth month of pregnancy the umbilicus commences to be less deep, by the seventh the depression is obliterated, and from this time onwards it protrudes more and more till it forms a rounded elevation on the abdominal wall.

During the last three months of pregnancy a physiological separation of the recti muscles occurs in the great majority of cases. The elasticity of the abdominal muscles is lessened, and when the distension is very great



an actual loss of tonus takes place. The muscles which chiefly suffer in this respect are the transversalis, the external and internal oblique muscles; while the recti, on account of their length and thickness, suffer to a much less extent, if at all.

*Changes in the Mammæ.*—From the second month onwards hypertrophic and other changes take place in the mammæ, resulting in gradual increase of not only glandular tissue, but of fat and connective tissue. The periphery of the breast is first affected. It becomes irregularly hard, and presents knotty masses which are enlarged acini of the glandular structures. Later similar hypertrophic changes spread along the ducts of the gland, towards the nipple, till ultimately the whole organ is affected. The distension of the skin leads to the formation of striæ similar to those seen on the abdominal wall, and through the tightly-drawn skin a network of veins can be seen, especially at the margins of the organs. The nipple becomes enlarged, more sensitive, and more readily erectile, and the pink areola surrounding it is replaced by an areola which varies in colour according to the complexion of the woman. In fair women it is of slight brownish colour, whereas in brunettes it is dark brown, or almost black. At the end of two months the areola is slightly raised, and the sebaceous follicles become more and more enlarged till they form distinct tubercles on the surface of the darkened area. These are known as Montgomery's tubercles. The usual diameter of the mammary areola is from one to one and a half inches; but in many cases, from the fifth or sixth month onwards, a "secondary areola" surrounding the primary is formed. This is not so deeply coloured, and is not always very marked. From the third month pressure on the gland causes the exudation of a thin fluid known as the colostrum; it increases in quantity as the gestation advances, and is the product of the newly-formed and of the developed pre-existing gland cells. It is an albuminous fluid, and contains fat cells, epithelial cells, and typical "colostrum corpuscles."

## II. GENERAL CHANGES

Pregnancy creates important changes in the different systems and organs of the body. The blood and the nervous system are affected to the greatest extent, but practically all the functions of the body are more or less modified. Since the pregnant woman has to perform nutritive and excretory functions for herself and her foetus, extensive changes in the general system must occur. So long as these changes take place within certain limits health is preserved and improved, but when carried to excess they lead to serious complications.

*The Toxæmia of Pregnancy.*—Much has of late been written on this subject. It is now believed by some authorities that many of the hitherto supposed physiological symptoms of pregnancy are the result of slight toxæmia, and that the graver complications of pregnancy, labour, and the puerperium are due to intense toxic infection. Vomiting, salivation, pigmentary changes in the skin, neuralgia, irritability of temper, etc., have generally been supposed to be normal since they are so common, but possibly they are due to the presence of toxins. As pregnancy advances, the general economy becomes adapted to the new conditions, and the symptoms of poisoning pass off. The emunctory organs work at higher pressure, but if one of them gives way, then symptoms of toxæmia become intensified, and may become so serious as to endanger the patient's life. Eclampsia is the most striking example of severe toxic infection. It is no longer regarded as a disease primarily of the kidneys. Post-mortem



examination reveals important changes in the liver similar to those produced by certain poisons. In many cases of eclampsia no pathological changes have been found in the kidneys, and the clinical evidence of disturbance of the renal function rapidly disappears after labour. The causes of these toxæmic conditions during pregnancy, labour, and the puerperium are varied, and include heredity, climate, previous renal disease, dyspepsia, hepatic disorders, anæmia, and tight lacing. Early recognition of toxic symptoms is important, as active treatment is frequently necessary to prevent serious complications.

*Changes in the Blood and Circulation.*—At one time it was supposed that the chief change in the blood during pregnancy was one of “plethora” of all its constituents, and “bleeding” was a very common operation during the later months of pregnancy. Now it is recognised that though the actual quantity of blood in the body is increased, the change in the nature of the blood is towards anæmia and hydræmia. The watery elements, the white blood corpuscles, and the fibrin are increased; but, on the other hand, the amount of albumin and of red blood corpuscles is diminished. The water increases from 791.1 per 1000, which is the normal amount in the non-pregnant condition, to 819.9 in the last month of utero-gestation. The red blood corpuscles diminish from 126 to 104.49. The albumin lessens from 70.5 to 66.1. The fibrin, which in the non-gravid state is 3 to 1000, lessens until the sixth month; then it begins to increase until, at the end of the ninth month, it is 4.3. The iron in the blood is necessarily diminished in amount, since the number of red cells is so decreased. There are, of course, great variations in individual cases. The hygienic surroundings and the nature of the food of the pregnant woman will materially affect the blood changes.

In 1828 Larcher first pointed out that a physiological hypertrophy of the heart took place during pregnancy. This increase in size is limited to the left ventricle and is such that the whole weight of the heart increases one-fifth during gestation. The increase in the total amount of blood in the body, the placental circulation, and the amount of abdominal pressure, all involve increased cardiac action, and this shows itself not in more rapid action of the organ, but in hypertrophy and in increased arterial tension. There is also during pregnancy a tendency to thrombotic and varicose condition of the veins. Durozier states that the heart continues enlarged during lactation, but if a woman does not nurse her child it diminishes in size immediately after labour. Women who have borne many children often have a permanent enlargement of the heart.

The liver is also enlarged and in women who have died suddenly after labour fatty degeneration has frequently been found. The *spleen* shows signs of marked hypertrophy, which is easily understood when one considers its important relation to the quantitative and qualitative changes in blood. It is increased in size from 140 grms. to 180 grms. The *thyroid* gland participates in the general change of increased growth, and is almost constantly somewhat enlarged during gestation.

*Changes in Respiration.*—Increase in size of the uterus mechanically interferes with the function of respiration. The long axis of the thorax is diminished by the upward displacement of the diaphragm, and this in the later months causes some embarrassment to breathing, but to a certain extent there is compensation in the fact that the lower thorax is increased in breadth. During the last two weeks of pregnancy breathing becomes easier owing to the “sinking” of the uterus and its contents. In the early months a certain amount of dyspnœa is occasionally observed, but this



is merely one of the numerous nervous reflex phenomena which so often complicate pregnancy. Since there is more blood in the body during gestation there is naturally an increase of the elimination of carbonic acid gas.

*Changes in the Digestive System and in Nutrition.*—In the early months of pregnancy the appetite is generally fitful, while nausea and vomiting within certain limits are so frequent as to be regarded as physiological. The varieties, causes, and significance of morning sickness are described under “The Diagnosis of Pregnancy” (p. 504). The appetite of the pregnant woman, even though often capricious to the very end of gestation, generally increases so that the general nutrition is improved. An increase in weight takes place irrespective of the enlargement of the uterus and the presence of a foetus. On an average the gain is from ten to fifteen pounds, or about one-thirteenth of the whole weight of the body. Excluding the generative organs the adipose tissue is increased more than any other tissue of the body. The deposits are specially marked in the mammary glands, in the abdominal walls, in the hips, and in the omentum. The whole figure therefore, becomes fuller and rounder.

The *temperature* of the body is not materially changed during pregnancy, although, according to some authorities, it is slightly lower in the morning than during the day.

*Changes in Bone.*—Besides the alterations in the bones and joints of the pelvis already referred to, changes in the bones of the skull have frequently been observed in women who have died during parturition. Rokitansky pointed out that irregular bony deposits were found on the under surface of the skull between the bone and the dura mater, and more especially developed on the parietal and frontal bones. These osteophytes consist of calcium carbonate, traces of phosphates, and organic matter. They have been discovered in rather more than one-half of all the cases examined of women dying after the fifth month. Some authorities consider that they are by no means peculiar to pregnancy, so that whether they are physiological or pathological has not been accurately determined.

During pregnancy there is always a risk of delayed union in fractured bones.

Owing to the growth and inclination forward of the growing uterus the centre of gravity is altered so that the pregnant woman is obliged to throw her head and shoulders backwards in order to preserve her equilibrium in the erect posture. The curves of the spine are increased, and this tends to an alteration in gait, which is especially marked in short women.

*Changes in the Skin.*—The pigmentation of the skin has already been referred to, but other changes are constant phenomena. The sebaceous and sweat glands acquire increased activity. Further, according to Barnes, “women who had been losing their hair when not pregnant found its growth restored during pregnancy, and falling out again after labour.” According to some authorities, the changes of pigmentation are associated with a temporary hypertrophy of the suprarenal capsules.

*Changes in the Nervous System.*—During pregnancy the nervous system becomes especially sensitive, and hence numerous reflex phenomena are observed. Rigors, flushings, fainting, hysterical disorders, disturbance of the special senses, and neuralgia are frequent. In the majority the mental state is one of despondency, especially in the early months, but often as gestation advances towards completion this state gives place to one of “buoyancy and a sense of joyful expectation.” It is quite the exception to meet with women who feel unusually well and cheerful throughout the whole pregnancy. Most look forward with great anxiety to labour, fearing



the worst for themselves and for their child. Changes in disposition are frequently very marked. A woman who is naturally amiable and bright may during pregnancy become moody and disagreeable, and the reverse is also sometimes observed.

*Changes in the Urine.*—The kidneys are somewhat enlarged and the urine is increased in quantity, but only in its watery elements. With the exception of the chlorides the solid constituents become less and less in amount as pregnancy advances. The phosphates and sulphates are supposed to be diminished owing to their being used in the growth of the foetus. At one time it was held that a white, grumous, soft “pellicle,” known as kiestein, was diagnostic of pregnancy. It forms in the urine about thirty-six hours after it is passed, and about the fifth day it breaks up and falls to the bottom of the glass; but it is now known that a similar substance is sometimes found in the urine of the non-pregnant and even in the urine of men.

During the last few weeks of pregnancy sugar may sometimes be detected in the urine, and during lactation it may be found in large quantities, diminishing and ultimately disappearing when the milk disappears from the mammæ. This temporary glycosuria must not be confounded with true diabetes, which is a serious complication of pregnancy.

Authorities differ as to the frequency of albuminuria during pregnancy. Schroeder maintains that in all pregnant women the urine contains albumin varying in amount from 3 to 5 per cent; others give the percentage as high as 20 or 30. No doubt albumin is detected in the urine of a very large number of pregnant women, and to many of these the albuminuria is physiological and transitory. This “temporary albuminuria” is much more marked during labour than during pregnancy, and the longer the labour the more likelihood of albuminuria. It is probably due to reflex vaso-motor spasm of the renal arteries resulting from uterine contractions.

The definite causes of albuminuria at any stage of pregnancy are really very uncertain, although many theories have been advanced to account for the condition. Increased intra-abdominal pressure, resulting in interference with the circulation in the vena cava and its branches, has been supposed to account for some cases, but this has never been proved. Most admit that the mechanical pressure of the gravid uterus on the renal vessels predisposes to the disease, and there are several facts which tend to uphold this theory. Albuminuria is more common in the later months of pregnancy than during the first five months. Those cases occur among primiparæ in whom the abdominal walls are tense and unyielding. Further, albuminuria is common in twin pregnancies, and when pregnancy is complicated by the presence of a fibroid or ovarian tumour. It is less frequent during pregnancy than during labour, when pressure is greater, and it diminishes after the birth of the child. The theory of increased intra-abdominal pressure is the oldest of all, but that this is sufficient to excite the disease in kidneys previously healthy has never been proved. It has been suggested that the albuminuria which sometimes occurs in the early months of pregnancy before there is any possible stagnation from pressure is purely reflex. It corresponds to the reflex irritation of the stomach which results in morning sickness. The hydræmic state of the blood is supposed to account for some cases of albuminuria, and in others the increased arterial tension may be the exciting factor. There is no doubt that in still others the albumin found in the urine of pregnant women is simply the result of vesical catarrh.

It is not necessary to do more than mention these cases in which preg-



nancy occurs in women suffering from chronic Bright's disease with albuminuria, and those who having previously suffered from kidney disease show albumin in the urine during pregnancy. These cases have quite a different relation to pregnancy from those already referred to.

The presence of albumin in the urine of a pregnant woman is always a cause of anxiety. If it is persistent and abundant, and especially if it occurs early in pregnancy, the outlook is grave, and careful treatment to prevent more serious complications is urgently required. Clinically, albuminuria suggests the possibility of uræmia and eclampsia.

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### Diagnosis of Pregnancy

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THE changes produced in the maternal organism by the condition of pregnancy are so very definite, and opportunities for observing them are presented so often to every medical man, that it might seem impossible for any one exercising due care to make a mistake when he is called upon to decide as to the presence or absence of pregnancy in a given case. That mistakes, however, are frequently made in this connection is a matter of common experience. Such mistakes are doubtless most often due to carelessness on the part of the practitioner—a too hasty examination of the patient, or a too hasty conclusion drawn from subjective phenomena only, without any physical examination. On the other hand, when it is remembered that Tardieu has said (and our knowledge has not advanced much since then) that except the bruit of the foetal heart, the signs of pregnancy, from the development of the abdomen and breasts up to the movement and efforts of labour, may be observed when there is no pregnancy, it will be seen that, even by the most skilful and careful observers, errors in diagnosis may occasionally be made.

The ridicule to which the author of such a mistake is apt to be subjected, and the loss of professional reputation which may result, would alone render this subject one of the utmost importance to every one engaged in the practice of medicine. There is, however, another and weightier reason. A wrong diagnosis may imperil the life of the patient. The treatment of certain diseases is very materially modified should pregnancy be also present. In the case of an ovarian tumour, which has been mistaken for a gravid uterus, complications may arise which make its removal both difficult and dangerous; on the other hand, a pregnant uterus which has been diagnosed as an ovarian tumour may be subjected to operation with fatal results, as has unfortunately not infrequently happened.



Whenever, therefore, the question of pregnancy arises, after the history of the case has been carefully searched a thorough physical examination of the patient should be made. Mistakes will then be rare. It is also a good rule to follow that, however strong the presumptive evidences of pregnancy may be, in the absence of one or more of the certain signs no absolutely definite opinion should be *expressed*. The lapse of a month, while it can but very rarely do harm, will generally be sufficient to clear up the diagnosis.

Pregnancy is revealed by certain symptoms and signs, subjective and objective phenomena. The importance of the latter over the former need hardly be referred to. A patient may not only be mistaken with regard to her sensations, but may purposely make erroneous statements about them. To the physician, therefore, the subjective phenomena afford at the most presumptive evidence only: the certain and infallible signs are perceived by his own senses.

SYMPTOMS.—*Subjective Phenomena of Pregnancy.*—I. *Suppression of Menses.*—Menstruation as a rule is in abeyance during the whole of pregnancy, and its stoppage is usually the first indication to the patient that she has conceived. So constant is this symptom that it forms the most reliable, and therefore generally adopted, datum from which to calculate the onset of labour. It must, however, be remembered: Firstly, pregnancy can occur before puberty, and also after the menopause. Secondly, menstruation may continue during the early months of gestation. Only in very rare cases is it met with after the third month, and most commonly it will occur during the first month only. In these cases the amount of blood lost is generally much less than the patient's normal. This, combined with the short duration of the flow, is often sufficient to arouse suspicion. Cases have, however, been recorded where menstruation went on regularly and normally all through the nine months of gestation, and Montgomery and Palmer record cases where it was never present except during pregnancy. Thirdly, that many other conditions—local, constitutional, climatic, and emotional—may cause amenorrhœa. In this connection the emotional or psychical causes of amenorrhœa call for special note.

Stoppage of menstruation is not infrequently met with in newly married women who are not pregnant but who are very desirous of becoming so, and who anxiously look forward to a failure of the menstrual flow as the first intimation that conception has occurred. More often it will be found in another class—generally unmarried women who have exposed themselves to the risk of impregnation, and are dreading the result. It is in these two classes of cases that mistakes in diagnosis are most easily made, because not infrequently when one such symptom appears, others, also psychical in their cause, are apt to follow, such as morning sickness and swelling of the breasts. To sum up, it may be stated that amenorrhœa, occurring in a healthy woman who has been previously regular, and who has been exposed to the chance of impregnation, is strong presumptive evidence of pregnancy, even in the early months; and its value increases, of course, the longer it continues. Confirmatory evidence must always be sought for, in the early months in the condition of the breasts, and in the later months in the presence of the abdominal tumour. An abdominal tumour associated with amenorrhœa, and whose size corresponds to the number of periods passed, should in every case excite the suspicion of pregnancy.

II. *Morning Sickness.*—Nausea and vomiting are an almost constant symptom in early pregnancy. It occurs, as a rule, in the morning on first



rising from the recumbent posture, and hence is familiarly known as morning sickness. In some cases, however, it may occur only in the afternoon or evening. This sickness is generally most severe in primiparæ and in highly-strung nervous women. It may come on immediately after conception, more often shows itself for the first time after the first menstrual suppression, and generally ceases during the fourth month. There may be merely a feeling of nausea, but this is usually accompanied by the vomiting of a glairy mucus. Lasting in most cases only a short time during the day, it causes, as a rule, no impairment of health. In some cases, when it becomes exaggerated and no food is retained, it may constitute a very serious danger (see "*Hyperemesis Gravidarum*"). The vomiting of pregnancy is generally considered to be a reflex disorder reflected from the uterus, and from its occurrence so very early in some cases this view is probably correct. The severer forms of the disorder may, however, be a manifestation of a toxæmia dependent in some way, not yet ascertained, on the pregnant state.

Taken by itself morning sickness is not a symptom of much value in the diagnosis of pregnancy, as vomiting in the morning after rising is not infrequently met with in ordinary gastric catarrh and in chlorosis. When it is associated with amenorrhœa and an unimpaired appetite it is significant presumptive evidence. It is valuable as a corroborative sign because it occurs so early in pregnancy.

Increased salivation is often found accompanying the severe forms of morning sickness. It may be present, however, alone. It is so rarely met with, however, as to be of very slight diagnostic importance.

III. *Various other nervous phenomena* which are occasionally met with in pregnancy—such as abnormal cravings for certain articles of food, changes in temperament (irritability or despondency), and neuralgias—are of no value in themselves as indicating pregnancy. When, however, any of them have been noticed in a patient in a former pregnancy they possess a certain significance.

IV. *Quickening*.—Much more important is the subjective phenomena of quickening, a term used to designate the sensation for the first time by the mother of the movements of the foetus *in utero*. The date of this is generally about mid-term, when the growing uterus comes in contact with the abdominal wall. As a sign of pregnancy it is obviously of much greater value in multiparous than primiparous women. The movement of flatus in the intestines and irregular contractions of the abdominal muscles may be mistaken even by multiparæ for quickening. Statements regarding the sensation of quickening must, therefore, be accepted with caution, and too great reliance must not be placed on them. If the foetal movements have not been felt by the mother this must not be taken as proof of the non-existence of pregnancy, for they are sometimes not noticed until very late, sometimes not at all even when the child is quite healthy; and, lastly, they will be absent, of course, when the foetus is dead.

V. *Bladder Irritation*.—Frequently during the first three months, while the uterus is still a pelvic organ, patients complain of some bladder irritation. Generally this is evidenced by frequent calls to micturition, in some cases by actual pain during the act. This is due to the pressure of the anteflexed uterus against the bladder, and passes off as soon as the uterus rises out of the pelvis. By itself this symptom is, of course, of no value, and even along with others is of little importance in diagnosis.

Kiestein, the flocculent deposit sometimes present on the surface of the urine in pregnancy, has no diagnostic significance as was once supposed.



It is composed of fungi mixed with triple phosphates. It is found also in the urine of non-pregnant women and also in that of males.

THE OBJECTIVE SIGNS OF PREGNANCY.—These, as already stated, are of much greater diagnostic value than the subjective symptoms just mentioned. These signs are made known to us by direct physical examination of the patient. This includes an examination of the mammæ, abdomen, vagina, and uterus.

CHANGES IN THE MAMMÆ.—These have already been considered under the head of local changes in the maternal organism resulting from pregnancy. We need, therefore, refer only to their relative value in diagnosis.

Montgomery regarded these changes to be *certain* signs of pregnancy if they were well marked. As a general rule this may be taken to be true if they are found present in a woman who is known not to have been previously pregnant. In parous women, however, their value is much diminished by the fact that in them the darkening of the areola, the prominence of the areolar tubercles, and secretion may be found present for years after lactation has ceased. They have been found present also in non-pregnant girls, it is stated, as a result of repeated sexual intercourse and mechanical irritation of the breasts and genitals. Almost identical changes, too, it must be kept in mind, are found occasionally in connection with various pelvic disorders, such as ovarian and fibroid tumours, chronic metritis, etc. The chief value of the mammary changes to the physician lies in the fact that they occur so early, and also because they can be so easily examined in a suspected case of pregnancy without exciting any suspicion in the mind of the patient as to the reason of the examination.

ABDOMINAL SIGNS OF PREGNANCY.—The changes in the size, shape, and appearance of the abdomen have already been stated. There are, however, other and more reliable signs. They are as follows:—

(a) *Active Movements of the Fœtus*.—These can generally be both seen and felt through the abdominal wall after the sixth month. In a multiparous patient they may be felt even much earlier. As seen these active movements must be differentiated from intestinal peristaltic movements. When felt in abdominal palpation they can hardly be mistaken for anything else.

(b) *Passive Movements of the Fœtus, Ballottement*.—The sensation which is imparted to the examining hand when the fœtus is moved about in the liquor amnii is called ballottement. It can be felt on abdominal examination if the fœtus is not too large, or the liquor amnii not too scanty, and is therefore most easily recognised about the sixth month. It is, however, subject to certain fallacies. A solid part of an ovarian tumour, when moved between the two hands, may impart a sensation similar to that of the fœtus in the liquor amnii. As practised in vaginal examination, when the patient's shoulders are raised, ballottement is one of the most reliable signs of pregnancy that we possess. It can generally be felt from the fifth to the eighth month, and can be simulated by nothing else. Vaginal ballottement is of special value in the diagnosis of hydramnios, when many of the ordinary signs of pregnancy are observed. In these cases vaginal ballottement is most clearly made out. It is of value also because it can be practised when the fœtus is dead if the liquor amnii has not escaped. It should be noted also that not only may a large fœtus and absence or deficiency of the liquor amnii obscure this sign, but that a malpresentation, such as a breech or placenta prævia, may make its recognition difficult or impossible.



*Intermittent Contractions of Uterus.*—The consistence of the uterus during pregnancy as felt on abdominal or vaginal examination will be found to vary from time to time. This variation is due to intermittent contraction and relaxation of its muscular wall. The interval between the contractions varies from five to fifteen minutes, and the duration of a contraction is from three to five minutes. On bimanual examination they can be felt as early as the third month, and they continue throughout pregnancy. They are not absolutely positive signs of pregnancy, as they are occasionally felt in soft, rapidly-growing fibrous tumours, but so very rarely that their presence may be taken as very strong presumptive evidence. They continue though the foetus is dead.

*Fœtal Heart Sounds.*—Mayor of Geneva in 1816 first pointed out that on auscultating the abdomen of a pregnant woman after mid-term the sounds of the foetal heart could be heard. In rare cases they may be recognised as early as the eighteenth week. They are generally compared to the sounds of a watch ticking under a pillow. The average frequency is from 135-140 per minute. They are less frequent in large than small children. The intensity of the sounds increases with the development of the child, but there is little change in their rapidity from the time they are first noticed till the end of pregnancy. The point of maximum intensity on the abdominal wall varies, of course, with the presentation and position of the foetus. Frankenhäuser's statement that the average rate of pulsation is greater in the female than the male is now considered to be erroneous.

*Uterine Souffle.*—A second sound heard from the fifth month onward is of the nature of a soft blowing murmur synchronous with the maternal pulse. It is due to the passage of the blood through the dilated and tortuous uterine arteries, and is therefore best heard over the sides of the uterus. The intensity varies very much in different cases, but it is to be heard almost constantly after the fifth month. As a similar bruit is found very often in fibrous and occasionally in ovarian tumours, the uterine bruit is not alone of great value in diagnosing pregnancy.

*Funic Souffle.*—A soft bruit synchronous with the *foetal* heart may also occasionally be heard. It is supposed to be due to some disturbance of the circulation in the cord produced by pressure.

THE CHANGES IN THE VAGINA IN PREGNANCY.—During early pregnancy, in the great majority of cases, the mucous membrane of the vulva and vagina is of a pale-violet colour, changing to deeper bluish tinge in the later months. It is noticed often as early as the second month, and is generally known as Jacquemin's test, and was first pointed out in 1837. The cause is a stasis of blood in the dilated vaginal blood-vessels. It is not a constant sign. Chadwick found it absent in 17 per cent of cases at the end of the third month. It is, however, usually present, and is a valuable corroborative sign in the early months when diagnosis is most difficult. Nor is it an infallible sign, for a similar discoloration is sometimes got in cases of fibroid and ovarian tumours, especially when these are impacted in the pelvis, and are obstructing the venous circulation in the pelvis.

*Softening and Enlargement of the Vaginal Portion of the Cervix.*—To the expert in vaginal examination these changes have a high diagnostic value from the third month onward. It has been stated that the absence of these changes in the cervix in a doubtful case of abdominal swelling reaching beyond the umbilicus may be taken as almost certain evidence of the non-existence of an intra-uterine pregnancy. The softening



of the cervix, which is got in the early stages of the cervical catarrh, and sometimes in intramural fibroid tumours, is not likely to be mistaken for the softening that occurs in pregnancy, except in the early months when it is not well marked.

*The Softening and Compressibility of the Lower Uterine Segment.*—This was first drawn attention to by Hegar in 1884, and is generally known as Hegar's sign. It can be noticed best if the lower part of the body of the uterus is grasped between two fingers,—one in the rectum, the other in the vagina,—when the difference between the comparatively hard cervix and upper part of the uterus on the one hand, and the soft lower uterine segment on the other, is felt to be very marked, and the compressibility of the lower uterine segment is easily made out. The softening and broadening out of the lower uterine segment can be made out also by simple vaginal examination through the anterior and posterior fornices. To the expert Hegar's sign is of great value in the diagnosis of early pregnancy.

*Changes in the Uterus.*—The changes in the size, form, position, and consistence of the uterus while it is still within the pelvis are the most reliable signs we possess for the diagnosis of early pregnancy.

Given favourable conditions for making a bimanual examination of the uterus in early pregnancy, *i.e.*, a lax abdominal wall and uterus in its normal position, the doughy softness, the fluctuation, the alternate relaxations and contractions of the pregnant uterus can hardly be mistaken for anything else.

Still, mistakes are made, and made frequently; therefore, while the uterine tumour is still within the pelvis, it is well not to be definite in an *expressed* opinion, at any rate, after a single examination.

#### THE DIFFERENTIAL DIAGNOSIS OF PREGNANCY

The increase in the size of the uterus being one of the most reliable signs in the diagnosis of early pregnancy, the conditions which are apt to be mistaken for it are naturally those which are also associated with this same sign. They are as follows:—

(a) *Chronic Metritis: Sub-involution.*—The uterus in metritis, though enlarged, will be felt to be uniformly hard, quite distinct from the doughy consistency of an early pregnancy. It is generally also associated with some degree of tenderness. Menstruation is profuse, and a history of some previous pelvic troubles will generally be obtainable.

(b) *A small interstitial fibroid* may be differentiated from pregnancy in the same way. It is much more difficult to differentiate between a small fibroid and chronic metritis than to distinguish between these conditions and pregnancy.

(c) *Enlargement of the uterus from retained menstrual or watery fluid* presents often much greater difficulties. *Retention of menstrual fluid* occurs in the great majority of cases in young girls in whom there is a congenital atresia of the cervix, vagina, or hymen. The uterine tumour which is found on abdominal palpation closely simulates, because of its doughy consistence, the pregnant uterus; but the fact that the patient has never menstruated, the pain which is generally present, and the absence of mammary signs, will generally excite suspicion and lead to a vaginal examination, when, if the atresia is in the hymen or vagina, the real nature of the case will be at once recognised.

Hæmatometron may, however, be got in women who have menstruated. The cause in them will generally be found to be an atresia of the cervix.



This atresia may be inflammatory in its origin, or be due to the application of escharotics to the cervix. A history of pain each month, the absence of mammary signs and ballottement, and a want of correspondence between the size of the tumour and the period of amenorrhœa as in the former case, will generally excite suspicion, though the use of a speculum or an anæsthetic may be necessary to enable us to make a positive diagnosis.

*Distension of the uterus* by a watery or mucous fluid is a rare condition. It is met with after the menopause, and is due to senile changes, producing an atresia of the cervix. Occurring as it does in old women it is not likely to be mistaken for a pregnancy.

In the differential diagnosis of these conditions, which may simulate pregnancy in the early months, it will be seen that we are largely dependent on the bimanual examination of the uterus. The necessity for caution in our expressed opinion at this period has already been stated. When from any reason the uterus cannot be satisfactorily palpated bimanually—it may be owing to the thickness or tension of the abdominal wall, or to a backward displacement of the uterus—there is still greater reason for being careful. Pregnancy in a retroverted uterus in the early months is very easily overlooked even by the most expert examiner.

From the fifth month onward pregnancy has to be differentiated from all those conditions which are accompanied by enlargement of the abdomen. The most important of these are—

*Ovarian Tumour.*—Cystic ovarian tumours, from their comparatively rapid growth, from their consistence, and from the fact that not infrequently they are associated with amenorrhœa, have frequently been mistaken for pregnancy. The converse is equally true. The following points should be remembered in differentiating the one from the other. Ovarian tumours are, as a rule, slower in growth than the pregnant uterus, and while in the latter the abdominal swelling is central from the first, in the case of the latter we can generally elicit a history that the swelling was first noticed at one or other side. If amenorrhœa is present with an ovarian tumour, it is very unlikely that the size of the abdominal swelling will have the same relation to the period of amenorrhœa as that of the pregnant uterus. Menstruation, however, is regular in the majority of cases of ovarian tumour found before the menopause. Secretion may be present in the breasts in cases of ovarian tumour, but only rarely is this so, and then the other mammary changes are absent. Examination in the case of ovarian tumours for the uterine bruit, foetal heart, and ballottement, will give negative results, and the uterus on vaginal examination can generally be felt not enlarged and displaced backward, forward, or to the side.

It is in cases of *hydramnios*, with consequent great abdominal enlargement, that mistakes are most apt to occur. Here several of the abdominal signs of pregnancy already mentioned may be absent, *e.g.* the foetal heart and uterine bruit, and the active and passive movements of the foetus. Vaginal ballottement, on the other hand, can be made out unusually distinctly, and is the most reliable sign we possess for the diagnosis of this condition. It may be noticed in passing that the sound may be passed into the pregnant uterus without rupturing the membrane, so that this simple operation must not be relied on to differentiate a pregnancy from an ovarian tumour.

*Fibroid Tumours of the Uterus.*—These, as a rule, are denser and grow more slowly than the pregnant uterus, and menorrhagia is generally a prominent symptom. In rare cases there may be found present mammary changes, but these are never so marked as those of pregnancy. A uterine



bruit is heard in more than half the number of cases, but the foetal heart sounds are of course absent, as are also the vaginal changes found in pregnancy. These points will generally serve to differentiate the one condition from the other. It must be kept in mind, however, that soft fibroids, and those undergoing cystic degeneration, may undergo a rapid increase in size, and that in soft fibroids the intermittent softening and hardening of the uterine wall so markedly felt in pregnancy may in rare cases also be noticed. In the absence of a very definite and reliable history it is very difficult to distinguish between a pregnancy in which the foetus has died and been retained *in utero*, and a fibroid tumour.

Hydatid degeneration of the chorion, producing an irregularly-shaped uterus and giving rise to irregular hæmorrhages, may closely simulate a soft fibroid tumour. The history, however, will be different in the two cases, and if this is not obtainable the lapse of a month will usually clear up the diagnosis.

Where a fibroid or an ovarian tumour and pregnancy co-exist the difficulty in diagnosis may be very great, but if the possibility of this occurrence is kept in mind, it will help occasionally in the diagnosis of an abdominal swelling with an obscure history and apparently contradictory symptoms.

*Distended Bladder.*—An over-distended bladder is not likely in itself to be mistaken for the pregnant uterus, though where it is present along with a pregnancy it may lead to certain errors in diagnosis. A distended bladder, for instance, the result of an impacted retroflexed gravid uterus, may be mistaken for hydramnios, or may cause one to suppose that the gestation is much further advanced than it really is. Such a mistake could only arise when a vaginal examination had not been made.

*Obesity.*—Accumulation of fat in the abdominal wall or in the omentum may not only render the diagnosis of pregnancy difficult, but may be mistaken for it. In women who are getting fat menstruation frequently becomes scanty and sometimes ceases altogether. This obesity is very often developed at the menopause. The increase in size of the abdomen and the cessation of menstruation naturally suggest pregnancy, but when other corroborative signs are sought for in the mammæ or vagina they will be found absent. Still, in stout women caution should be observed in expressing a negative opinion, for obesity obscures most of the abdominal signs of pregnancy, and also renders the vaginal examination very difficult.

*Spurious Pregnancy.*—This is a rare condition, met with usually in neurotic patients, or in women who have married late in life and are desirous of becoming pregnant. In it all the symptoms and several of the signs of a true pregnancy are present. Menstruation may stop, the breasts secrete milk and enlarge, and there is morning sickness. Later there is swelling of the abdomen, quickening is felt, and the patient passes into a spurious labour. If suspicion is aroused and a careful examination made, a mistake in diagnosis is not likely to be made. A resonant percussion note over the abdominal swelling alone excludes a true pregnancy. Should the patient be stout an anæsthetic may be necessary. In narcosis the abdominal tumour generally subsides.

*Tumours of other Abdominal Viscera: Liver, Spleen, Kidneys.*—These are not so likely to be mistaken for pregnancy as tumours of the ovary or uterus. Their growth is usually slow, and, with the exception of the abdominal swelling which they produce, they are unaccompanied by any of the other signs of pregnancy. The lower border of such tumours is free, and



the edge of the hand can be passed between it and the pubis, thus proving their abdominal and not pelvic origin.

*Ascites.*—Ascites could hardly be mistaken for pregnancy. Fluctuation is very distinct, the character of the percussion note changes with the position of the patient, and the signs of pregnancy are absent.

**THE DIAGNOSIS OF THE PROBABLE DATE OF CONFINEMENT.**—The date of delivery can be predicted only approximately, firstly, because the duration of human gestation varies within certain limits, and, secondly, because even if this duration was constant, we are unable to determine exactly when fertilisation of the ovum takes place. We do know now that ovulation occurs irregularly, and not only in relation to menstruation as was formerly supposed, and from observations in the lower animals it is probable that an interval of from one to fourteen days may elapse between insemination and fertilisation. So far as we can determine, the average duration of human pregnancy is from 270-280 days. It is found also that the first week immediately following menstruation is the most favourable time for fertilisation, and this fact is made use of in calculating the date of the onset of labour. Naegle's method, because of its simplicity, is the one generally used. It consists in going back three months from the date of the beginning of the last menstruation and adding seven days; *e.g.* the date of the commencement of the last menstruation being August 7, labour may be expected about the 14th of the following May. Where the menstrual history of the patient is available no more accurate method can be got; but an error of from one to three weeks even with this is possible. The possibility of menstruation in the early months of pregnancy must always be borne in mind. Very often, however, as in cases where menstruation is irregular or absent, as in lactation, anæmia, phthisis, before puberty, etc., this method of calculation is impossible.

*The Date of Fruitful Coitus.*—This datum is rarely available in married women, though some profess to know from their sensations during coitus if conception has taken place. It is useful, however, in those cases where there is a history of a single coitus. The onset of labour may then be predicted at the 275th day after coitus.

*The Date of Quickening.*—Quickening generally occurs about the third term of pregnancy, and may therefore be used, in the absence of more reliable data, in calculating the probable date of confinement. In an intelligent multiparous woman it is of distinct value for this purpose. In all cases it is useful as a check where the menstrual history is indefinite, and is probably the most reliable datum available where from any cause menstruation has been suspended before impregnation.

*The Height of the Fundus Uteri.*—This depends to a considerable extent on the amount of liquor amnii and the size of the fœtus. Roughly speaking, however, it may be said that the fundus can be felt just above the symphysis pubis in the fourth month, at the fifth month midway between the symphysis and umbilicus, at the sixth month at the level of the umbilicus, at the seventh month two finger-breadths above the umbilicus, at the eighth midway between the umbilicus and the ensiform cartilage, at the ninth it reaches the ensiform, and in the tenth month the fundus sinks somewhat to a lower level. The level of the fundus, in many cases, does not vary much in the last weeks. The condition of the cervix at this time, however, will enable us to tell at least if labour is imminent, as it undergoes a *real* shortening, and its canal becomes merged into that of the uterus in the last fortnight of pregnancy.

*The Length of the Fœtus in Utero.*—This, according to Carl Braun,



forms one of the best data from which to estimate the time of pregnancy. As it lies folded up in the uterus, the length of the foetal axis is found to be about one inch for every lunar month of gestation. The measurement is got by applying one arm of a pair of callipers to the lowest part of the head, the other to the highest part of the breech. In primiparæ, where the head is already within the brim, it is necessary to introduce the lower blade into the vagina. A measurement of seven inches got in this way would indicate a seven months' gestation. This method of calculation is not, however, likely to be much used in general practice.

In calculating the onset of labour we will then generally rely on Naegle's method. It is well never to be too definite, a considerable margin should always be allowed, as mistakes of from one to three weeks are common.

DIAGNOSIS OF THE DEATH OF THE FŒTUS.—The indications of intra-uterine death may be briefly stated as follows:—

- 1. The disappearance of the foetal heart sounds if these have been previously noticed.
- 2. The cessation of active foetal movements.
- 3. A slow diminution in the size of the abdominal tumour.
- 4. Diminution in size and tension of the mammae.
- 5. Symptoms on the part of the mother of malaise. Chilliness and a feeling of dead weight in the lower abdomen.
- 6. Fœtid discharge from the uterus if the membranes have been ruptured.
- 7. Looseness and mobility of the foetal cranial bones and feet on vaginal examination if the finger can be introduced within the internal os.

If the membranes remain unruptured the liquor amnii is slowly absorbed, and the dead foetus may be retained *in utero* for weeks and even months. Such a consideration, as already stated, in the absence of a definite and reliable history, may present great difficulty in diagnosis.

THE DIAGNOSIS OF PRIMIPARITY AND MULTIPARITY.—Under certain circumstances, generally medico-legal, it may be necessary to determine if a woman is pregnant for the first time or has previously been so. Traces of a former pregnancy which has gone to full time or past the sixth month can generally be found in the lax and pendulous condition of the mammae, the relaxed abdominal wall, the lineæ albicantes, the absence of the hymen, the patulous condition of the vagina and vaginal orifice, and the lacerated cervix. A pregnancy, however, which has terminated in an abortion before the sixth month may leave no traces at all by which it can be subsequently recognised. It must also always be kept in mind that the lapse of a few years may obliterate many of the above signs, and that certain pathological conditions of the cervix in a primipara may closely simulate the characters of the cervix in a multiparous woman.

Diagnosis of Multiple Pregnancy. (See "TWINS.")

Multiple Pregnancy

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1. *DEFINITION and Varieties.*—One child at a birth is the natural or normal law in woman, but plural conceptions are not infrequent. The

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pregnancy is then spoken of as multiple. Two at a birth is comparatively common as compared with higher numbers, and is taken as the type of plural conceptions. Cases of three are rare, once in about 7000 deliveries; four still more rare, and of five not more than six or seven cases have been recorded. More than five at a birth has not been reliably authenticated.

2. *Frequency*.—The proportion of twins to single births may be regarded generally as one in 80 deliveries. Variations in frequency are shown in the statistics of different countries, from 1 in 60 (Bohemia and Ireland) to 1 in 100 (France), or 1 in 120 (New York). These apparent differences, however, may, to a large extent, be due to different systems of registration, and to defects, such as omission of abortions in the registers. The frequency is no indication of the comparative fertility of the respective nations.

3. *The influences which predispose to twin-bearing* have attracted considerable attention, but the conclusions arrived at reach only a varying degree of probability. There is abundant evidence to show in many cases a *hereditary* influence, and also to support the opinion that there is an association between *twin-bearing and prolificacy*, but these influences, in all probability, apply to one class of twins only, the binovular, and not to the monochorionic or homologous twins.

The influence of the mother's *age and the number of the pregnancy* have been carefully worked out by Dr. Matthews Duncan. The following statements indicate the results of his investigations regarding the points in question:—

(1) The mean age of twin-bearing mothers is greater than that of mothers generally.

(2) While of all births three-fifths occurred among women under thirty, there occurred only two-fifths of the twins among the younger women.

(3) From the earliest child-bearing period till the age of forty is reached, the tendency to twin-bearing increases—or, in other words, the proportion of mothers of twins to mothers of the same age increases as the fertile women grow older.

(4) The same is found as regards the initial fertility. Newly married women are more likely to have twins the older they are. Every one hundred and fifty-third woman among the youngest fertile women bears twins within two years after marriage; among the older women, from thirty-five to forty years of age, every forty-second woman bears twins within two years after marriage, or nearly four times as many.

(5) As regards the number of the mother's pregnancy, the proportional frequency of twins increases with the number of the pregnancy. To this general statement there is an exception in the case of first pregnancies. Woman has apparently an increased chance of bearing twins in her first pregnancy.

*Influence of the Male*.—Cases have been recorded which appear to support the view that the male may exert some influence in the procreation of twins; as where a man twice married has had twins by both his wives; or where a marked hereditary tendency to twin-bearing has been traced in successive generations, it has manifested itself in the family of one of the males. Seeing, however, that twins must primarily depend on ovulation, such instances may be regarded as mere coincidents, unless it can be shown that the generative vigour of the husband can stimulate the function of ovulation in the wife.

4. *Origin and Development*.—Twins may be developed from two ova, *binovular*, derived from either a single Graafian follicle, or two separate ones, in the same or different ovaries; or they may take origin from one ovum, *uniovular*, containing a double germ.

Ahlfeld has advanced the view that twins may take origin from division in the formative material within a single area *germinativa*.



When the division is complete separate twins are produced within a single amnion, or if incomplete, conjoined twins or some form of double monster is the result.

Evidence of the origin and mode of development can be obtained by making a careful examination of the placenta and membranes. This should always be done, especially where anything peculiar in the foetuses or labour has been observed. The want of any note as to the relation of the membranes to the foetuses has much impaired the value of the record of cases. In certain conditions the relationship may be difficult to determine. The separability of the chorion from the amnion at full term and the difference in transparency make easy the recognition of the envelopes. During examination the structures should be placed under water.

When *binovular* there are *two chorions, dichorionic*, and *two amniotic sacs*, and if the ova have been embedded in the mucosa sufficiently apart, each will have had a separate decidua reflexa, but when close together the latter may have been in common.

When *uniovular* the chorion is single, *monochorionic*, but with two amniotic sacs in nearly every case. Occasionally both foetuses lie in a common amnion. A single chorion occurs once in eight cases, a single amnion once in 132 twin pregnancies (Ahlfeld).

5. *Examination of Placenta and Membranes.*—*a.* When the placenta are separate, and each foetus lies in its own chorion and amnion, there can be no doubt of their origin from two distinct ova. Although developed separately the placenta, in the process of growth, may come in contact and so combine as to give the appearance of a common placenta. The double chorion will indicate the distinct origin. In such cases the placental vessels are entirely distinct, there is no anastomosis.

*b.* If only one chorionic covering is present the foetuses have been derived from a single ovum with two germs. In such cases the placenta are always united, and it is not rare for anastomosis to have existed between the two circulations. The cords may be inserted close together or widely apart.

*c.* In such cases, with a single chorion, there is nearly always two amnions. Both foetuses, however, may be found to have occupied a common amnion. The sac may have been single from the first, for the blastoderm may envelop both foetuses in a common amnion; but in other cases it would appear, that though primarily double, the cavities have been reduced to one by the absorption of the septum or portion of the two sacs in contact. Remnants of the septum have been found, and traces may be detected, between the insertions of the cords, in the forms of lines of fat from degeneration of the tissues and adhesion of the amnion beneath.

*Homologous Twins.*—This term has been applied by Ahlfeld where normally formed twins of the same sex have been enclosed within one chorion and drawn their nourishment from a single placenta—the *monochorionic disomata* of teratologists. They are strikingly alike, and present great similarity in bodily development and even in disease.

A very different result is found in another group of this class, the *allantoido-angiopagus* (Ballantyne) or *omphalo-angiopagus* of other writers. They are defined as “twins of the same sex, enclosed within a single chorion, one of which shows more or less grave developmental defects, and communicates with the other by means of the vessels of the umbilical cord in the neighbourhood.” The modes by which this anastomosis occurs are very numerous, they are both within and without the placenta, and it thus becomes possible that the circulation of the deformed twin, which may not have a heart, is maintained by the heart of the normal twin.



Hence the importance of carefully describing the vascular arrangement in all cases of twin monstrosities, and of including the placenta and membranes when sending a specimen of deformity to a specialist.

6. *Sex of Twins*.—Monochorionic twins are always of the same sex. When the chorion is double they may be of the same or different sex. Males preponderate over females, but to a lesser extent than in births generally. Most frequently the two children are of different sex. In the Prussian statistics, in 150,000 twin pregnancies, a boy and girl occurred 54,000 times, both males 50,000, and both girls 46,000 times.

7. *The Diagnosis*.—The presence of more than one foetus *in utero* may at times be suspected from the size and form of the uterine tumour, but a certain diagnosis can be made before labour only on the evidence of palpation and auscultation. The uterus need not necessarily be large if the twins are small, or there is great disparity in size; and a large uterus may be due to an extra-sized single foetus, or to an excess of liquor amnii. The evidence of a large quantity of the waters, on the other hand, does not negative twins, for that condition is not infrequently present in plural pregnancies. In the earlier months there is no certain sign. When pregnancy is sufficiently advanced to permit of the foetal parts being recognised by palpation, the definite character of a *head* may be made out *in two places*, but the apparently numerous points of *foetal limbs* are liable by themselves to deceive. The *foetal heart sounds* heard readily in two places are insufficient; but when two points of greatest intensity are found at some distance and on different sides of the uterus, with a line of diminishing intensity and again increasing between, or the sounds lost and again found, the inference of plural pregnancy is very strong, and is made certain if on two or three observations made simultaneously by two observers, the rate of the heart-beats proves to be different at the two spots.

8. *The course of pregnancy* is more liable to be disturbed when plural than when single. Abortions are comparatively more frequent, and there is an increased tendency for labour to ensue more or less prematurely. This does not arise, as is generally supposed, from the over-distension and greater irritability of the uterus, but from the feebler organisation and consequent earlier occurrence of the physiological degenerative changes which are now known to precede the onset of labour.

When the foetuses are developed from two distinct ova, *dichorionic*, the prospects are much more favourable. Inequality in development and growth is common—one foetus, as a rule, is more favoured than the other. The relative position in the uterus is an insufficient explanation, for the most favoured is not always the first to be born. In the *monochorionic* variety defective development and nutrition are more likely to occur, and to lead to malformations or death of one of the foetuses. The degree of proximity of one embryo to the other, their relation to the decidua serotina, and the union of the two allantoides, are the chief factors in causing interference. The greatest diversity is found where both foetuses derive their nourishment from one common placenta.

One foetus may die and be retained without undergoing maceration or decomposition. It is expelled and found amongst the membranes, flattened and shrivelled, the *foetus papyraceus v. compressus*.

Well-authenticated cases have been recorded where one foetus has aborted and been expelled, the pregnancy, nevertheless, continuing with the other and reaching maturity at the full time.

In the later months a considerable interval of days and even weeks in a few cases has elapsed between the births of the two children. Some



of these have been proved to be due to *uterus duplex*, and probably all are. Such an occurrence, and the occasional great disparity between the two children, gave origin to the hypothesis of *superfœtation*, by which is meant the possibility of a woman being capable of conceiving a second time when already pregnant for one or more months. Excluding ectopic gestation and the presence of a double uterus, such a hypothesis is now held to be "physiologically untenable." *Superfecundation*, or the impregnation of a woman a second time, by two separate sexual acts within one intermenstrual period, is totally different, and has been proved to occur by cases where the fathers of the twins have been of different races. But the short interval within which such second impregnation must take place does not explain the great disparity in development, nor the greater interval that has occurred in some cases between the separate births.

### Management of Pregnancy.

Pregnancy, besides producing local changes in the sexual apparatus, is the cause of a widespread alteration throughout the whole of the body. The organs of a pregnant woman have to adapt themselves to a changed order of things (see "Physiology of Pregnancy"). Possibly the most important change is the conspicuous alteration in the quality of the blood and the increase in its quantity, this throwing an increased strain on all the excretory functions—lungs, kidneys, bowels, skin. If any of these organs are not equal to the increased pressure put upon them, symptoms, moderate or severe, of toxæmia supervene (p. 499).

The main indication for management thus lies along the lines of maintaining as good a hygienic state as possible, and when any organ is known not to be quite sound, to assist it as far as possible to stand the extra strain.

When a pregnant woman is healthy and leads at ordinary times a sensible hygienic life, nothing is better than that she should adhere to the same mode of life as suited her well before.

*Regular exercise* in the open air is most important, and should be insisted upon. It is undoubtedly injurious to lie in bed most of the forenoon, and rest on a sofa in the afternoon. Such practices are only justifiable in a healthy woman, at the date at which a menstruate period is due, if there is a history of previous abortions. This "over taking care" brings on indigestion and sleeplessness, and also loss of tone in the muscles, which makes the labour more prolonged, and brings about a loss of recuperative power during the puerperium. We have a good object-lesson in the wives of working men, who continue to work until labour commences, without detriment to themselves.

Violent exercise of any sort tends to produce miscarriage. Driving over rough roads, prolonged railway journeys, dancing, and bicycling, except in the early months, should be forbidden. The lifting and carrying of heavy weights must be avoided; while the continuous use of a sewing-machine or systematic massage are also not advisable.

The increased elimination of carbonic acid gas makes it imperative that the patient should have plenty of fresh air; thus crowded churches, theatres, concerts, balls, and crowded, stuffy rooms should be avoided. Going to bed early and sleeping in a large, well-ventilated room are important. Mental excitement should be avoided. Irritability of temper requires great forbearance on the part of the patient's friends.



*Clothing.*—The enlargement of the breasts and abdomen and the increase of the cutaneous vascular system make it necessary to have a change in the clothing. Light warm garments to prevent chilling of the body, and roomy clothes that do not interfere with the respiration and circulation, are required. The dress should be loose and easy, with no tight waistband. After the three and a half months the ordinary corset should be discarded and special corsets used, which support the enlarging breasts without pressure on the nipple, and which also have a band at the lower edge which can be let out on either side with laces as the abdomen increases in size. In multiparæ with flaccid abdominal walls, a belt will be found of great comfort; it can be made of flannel or lint, and made to fit.

Tight garters are injurious, stocking suspenders being used instead; the sooner these are used the better chance of avoiding troublesome varicose veins. After the fifth month, when the increasing abdomen lifts the petticoats off the lower part of the abdomen and thighs, loose flannel drawers should be worn to prevent chills.

*Diet.*—As far as our knowledge at present takes us, the diet of a pregnant woman should be a good general mixed diet of plain, well-cooked, simple food, taken at regular intervals. The quantity of nourishment need not exceed what is usually required by a healthy woman, but it is well to take food in smaller quantities and more frequently during the later months, when the enlarged uterus presses against the stomach. “Longings,” so far as they are kept within reasonable desires, can, of course, be gratified; but no evidence is obtainable that the refusal to satisfy unreasonable caprices has any influence mentally or physically on the child. Strong alcoholic and malted drinks are unadvisable, but there is no reason to prohibit a little light wine at dinner and lunch, if such has been the habit of the patient previously.

That the diet must directly influence the growth and development of the foetus *in utero* is reasonably clear. Recent scientific experiments seem to show that possibly at no very distant date we may learn in what manner the growth and development of the bones of the foetus and the tissues of the mother may be favourably modified by a special dietary, so that labour may be shortened and made more easy for the mother, and less dangerous to the child. Indeed there are indications that diet may so influence the maternal and foetal tissues, so as to lead to the birth of a viable child in cases where the previous obstetric history indicated the improbability of this ever taking place.

Two suggestive cases have come under the writer’s notice. The first was a multipara who had had several confinements, some of them premature, but never a living child, owing to the size of the pelvis. When I saw her she was on a vegetarian diet. The labour came on prematurely at the eighth month, and she was easily delivered of a healthy infant which lived. The second case was a primigravida, æt. about twenty-six, a very small woman, who was a rigid vegetarian (from conviction); she was in the second stage of labour before feeling anything, and the infant was born very rapidly, certainly an exceedingly easy labour for a primipara of her age. It is, of course, possible, but I think unlikely, that these were merely coincidences.

It is probable that the diet which will ultimately be found to exert a beneficial influence in this direction will be one in which the carbohydrates and fats enter most largely. If space allowed, many facts could be cited from comparative obstetrics, and also from the obstetric histories of different races, in favour of this view. With our present knowledge it is sufficient to suggest that this line of investigation should be studied



carefully from the clinical side no less than from the standpoint of experimental laboratory observation.

THE REGULAR EVACUATION OF THE BOWELS is very important; during pregnancy there is always a tendency to more or less constipation. This difficulty is best got over by giving a liberal supply of fruit. If medicine is necessary, some of the mild vegetable purgatives are to be recommended, cascara sagrada, liquorice powder, or tamar indica lozenges. The constant use of mineral waters tends to aggravate hæmorrhoids, which are often very troublesome, and strong cathartics are not safe as they may be the cause of a miscarriage. Castor-oil is a safe but to most people a very disagreeable remedy. Attention to this point is probably the most important factor in the prevention of many of the minor and even major manifestations of toxæmia in pregnancy.

During the early months complaints of inability to pass water, or constant desire to micturate, should put the practitioner on his guard for cases of retroversion. The urine should be examined from time to time, especially in the later months, and more particularly in primiparæ. If albumen be detected, special precautions must be adopted (see "Pregnancy, Pathology of").

The *skin* requires special care. It should be kept in good condition by frequent bathing. When acting properly it is able to relieve the kidneys of a portion of the extra strain thrown on them. Women who are in the habit of taking a daily cold bath should continue to do so, and should take in addition a warm bath once a week (90°-96° F.); those who are not accustomed to the daily bath should have a tepid bath twice a week (77°-90° F.). During the last six weeks the warm baths may be taken three or four times a week; this is specially good for primigravidæ.

The increased vaginal secretion renders it important for the external genitals to be frequently cleansed. These can be washed morning and night with tepid water and a good lather of soap, a little spirit and water rubbed in afterwards; an advantage, as it prevents any heavy odour. If the vaginal discharge becomes irritating, either causing pruritus or excoriations, the vaginal douche may be used. The quantity injected should not exceed a pint of water, should be injected very slowly, and only lukewarm water used. Chafing under the breasts and groin can be got rid of with washing twice daily with vinegar and water, drying carefully, and dusting on boracic acid.

The *breasts* should be kept warm and supported without any undue pressure upon them. The nipples and areola during the last two months should be hardened by regular washing with water and alcohol. The methods of bringing forward depressed nipples, such as pressing back the areola with elastic rings or a nipple shield, or pulling forward the nipple, usually give a good deal of pain, and it is doubtful if they do any good.

Sexual excess in young women is often the cause of early abortions, and abstinence is advisable.

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